MEDICAL PROCEEDINGS

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REDAKSIONEEL · EDITORIAL

POLIOVIRUS, FORMALDEHIED EN DIE WET

Ongeveer drie jaar gelede, nadat Amerikaanse deskundiges aangekondig het dat die Salk-poliomiëlitisenstof veilig en doeltreffend is, het etlike vooraanstaande farmaseutiese firmas, almal waarvan 'n vername posisie op die gebied van navorsing en die bemarking van biologiese preparate beklee het, tot die uitgebreide kommersiële produksie van die entstof in die Verenigde State oorgegaan. Daar sal herinner word dat binne 2 weke of wat ongeveer 79 kinders poliomiëlitis opgedoen het nadat hulle ingeënt is met entstof wat deur die Cutter-laboratorium geproduseer is. Lewendige virus is in werklikheid gevind in 6 van die 17 Cutter-klompe wat ondersoek is. Die Openbare Gesondheidsdiens in die Verenigde State het destyds besluit dat die poliomiëlitis wat deur die betrokke kinders opgedoen is, aan die toediening van die Cutter-entstof gewyt moes word, en strenger toetsmetodes is onmiddellik ingestel.

Die verhaal het nie geëindig by diegene wat ingeënt is nie; 125 persone wat in noue aanraking gekom het met die ingeënte kinders het ook poliomiëlitis opgedoen. Altesaam was daar 11 sterfgevalle.

Hierdie treurige ramp het in Januarie vandeesjaar 'n nasleep in die Amerikaanse hof gehad toe 'n jurie bestaande uit 8 vroue en 4 manne 27 dae lank getuienis oor die beweerde aanspreeklikheid van die Cutter-organisasie aangehoor het.

Die sake wat in die hof gedien het, was inderdaad toetssake. In die een geval is daar 'n eis om $300,000 ingestel, en in die ander

POLIOVIRUS, FORMALDEHYDE AND THE LAW

Some three years ago, after the American experts had pronounced the Salk poliomyelitis vaccine safe and effective, extensive commercial production of the vaccine was undertaken in the U.S.A. by several prominent pharmaceutical firms, all of whom held distinguished positions in connexion with research into and the marketing of biological preparations. It will be recalled that within a matter of 2 weeks or so, some 79 children contracted poliomyelitis after having been inoculated with vaccine produced by the Cutter Laboratories. Live virus was actually found in 6 of 17 Cutter batches examined. At the time the United States Public Health Service concluded that the poliomyelitis in these cases resulted from the administration of the Cutter vaccine. More stringent test methods were introduced immediately.

The tale did not end with those inoculated; 125 close contacts of the vaccinees also contracted poliomyelitis. There were, in all, 11 deaths.

This distressing catastrophe had its sequel in the American courts in January this year when a jury of 8 women and 4 men heard evidence for 27 days on the alleged liability of the Cutter organization.

The actions being tried in court were, in fact, test cases. In one the claim was for $300,000 and in the other for $65,000. The jury found that the poliomyelitis was caused by the Cutter vaccine and that there was live virus present in that vaccine. The issue of negligence, however, resulted in a formidable

om $65,000. Die jurie het bevind dat die poliomiëlitis veroorsaak is deur die Cutter-entstof, en dat daar lewendige virus in die entstof was. Wat die kwessie van nalatigheid betref, was daar egter 'n hewige meningsverskil tussen dr. Jonas E. Salk (wat getuienis vir die eiser afgelê het) en dr. Wendell M. Stanley (aan wie 'n Nobel-prys toegeken is vir sy oorspronklike werk op die gebied van die kristallisasie van virusse). Dr. Salk het beweer dat formaldehied die poliovirus op 'n voorspelbare manier doodmaak ooreenkomstig 'n reglynige (logaritmiese) verhouding. Hy het volgehou dat na 'n paar dae lange inkubasie met formaldehied so min virus-deeltjies oorbly dat hulle nie maklik bemerkbaar is nie. Volgens sy teorie in verband met virusvernietiging, egter, het hy die mening uitgespreek dat die vernietiging van die virusse voortgesit is ooreenkomstig die grafiekverhouding wat hy voorspel het.

Dr. Stanley het geheel en al van dr. Salk verskil. Sy waarnemings het hom tot die gevolgtrekking laat geraak dat die kromming in verband met die onaktiefmaking van die poliovirus met formaldehied nie die hipotetiese reglynige verhouding van dr. Salk volg nie. Dr. Stanley het ook die mening uitgespreek dat die toetse wat destyds toegepas is, geheel en al ondoelmatig was in die lig van latere kennis—'n sienswyse wat die moontlikheid van nalatigheid aan die kant van die Cutter-organisasie heeltemal uitskakel.

Die jurie het blykbaar dr. Stanley se getuienis aanvaar en met 10 teen 2 beslis dat die Cutter-organisasie nie nalatig was nie in die lig van die wetenskaplike kennis wat beskikbaar was op die tydstip toe hulle hul produk voorberei en bemark het. Daar is egter beslis dat 'n stilswygende waarborg verstrek is dat iedere ampul entstof veilig was, en gevolglik is skadevergoeding tot 'n bedrag van $131,500 aan die eerste eiser, en $15,800 aan die tweede eiser toegeken.

Daar word verneem dat die Cutterorganisasie voornemens is om appèl teen hierdie uitspraak aan te teken. Die uitslag van die appèl sal 'n belangrike effek uitoefen op die ander eise namens beweerde slagoffers van die Cutter-entstof. Hierdie eise beloop altesaam $10,000,000.

Wat ook al die uiteindelike gevolg van hierdie regsgedinge gaan wees, is dit gerusstellend om daaraan te dink dat miljoene dosisse van die Amerikaanse entstof gedurende die afgelope 3 jaar uitgereik is sonder enige gevaar, en met oortuigende statistiese bewyse dat die entstof beskerming teen verlammende poliomiëlitis verleen. Die probleem bly natuurlik nie tot die vervaardiging van 'n veilige entstof beperk nie. Die entstof moet veilig sowel as antigenies en beskermend wees. Hierdie moeilikheid word geskep deur die fyn ewewig tussen algehele onaktiefmaking van die virus met daaruit voortspruitende antigeniese ondoeltreffendheid, en 'n mate van onaktiefmaking wat nie die beskermende antigenisiteit vernietig nie.

Die juiste werkwyse word nog nie volkome begryp nie. Poliomiëlitisvirusentstof is uniek onder die

controversy between Dr. Jonas E. Salk (who was called by the plaintiff's attorney) and Dr. Wendell M. Stanley (a Nobel prize winner for original work in crystallizing viruses). Dr. Salk maintained that formaldehyde killed poliovirus in a predictable manner according to a rectilinear (logarithmic) relationship. He argued that after incubation with the formaldehyde for a few days, so few virus particles were left as not to be easily detectable. According to his theory of virus destruction, however, he held that the viruses continued to be destroyed according to the graphic relationship he had predicated.

Dr. Stanley was completely at variance with Dr. Salk. His observations led him to the view that the curve relating to the inactivation of poliovirus by the formaldehyde does not follow Salk's hypothetical straight-line relationship. Dr. Stanley also expressed the view that the tests applied at the time were grossly inadequate, in the light of subsequent knowledge—a view which removed any possibility of negligence on the part of the Cutter organization.

The jury apparently accepted Dr. Stanley's evidence and voted (10 to 2) that the Cutter organization had not been negligent in the light of the scientific knowledge available at the time they prepared and marketed their product. As it was held that there was an implied warranty that every ampoule of the vaccine was safe, they awarded damages on the scale of $131,500 to the first plaintiff and $15,800 to the second plaintiff.

It is understood that the Cutter organization intends to appeal against this finding. The result of the appeal will affect other claims on behalf of alleged victims of the Cutter vaccine. These claims total about $10,000,000.

Whatever the eventual outcome of these proceedings, it is reassuring to reflect that millions of doses of American vaccine have been issued in the last 3 years without any danger and with powerful statistical evidence that the vaccine is protective against paralytic poliomyelitis. The problem, of course, does not lie in manufacturing a safe vaccine, but in producing one which is not only safe but also antigenic and protective. This difficulty is created by the nice balance between complete inactivation of the virus, with subsequent antigenic ineffectiveness and a degree of inactivation which will not destroy protective antigenicity.

The true mode of action is still not fully understood. Poliomyelitis virus vaccine is unique among virus vaccines in that it is apparently an inactivated preparation. Most other virus vac-

virusentstowwe omdat dit oënskynlik 'n onaktief-gemaakte preparaat is. Die meeste ander virusent-stowwe is biologies aktief, maar in 'n verswakte vorm. Trouens, die mening is reeds uitgespreek dat poliovirusentstof alleen doeltreffend is in die mate waarin 'n sekere klein aantal lewendige virusdeeltjies daarin slaag om die proses van onaktiefmaking te oorleef.

Maar wat die suiwer wetenskaplike posisie ook al is, is dit duidelik dat daar geen regverdiging is vir die versaking van die ingrypende toetsmetodes wat voorgeskryf is nie, en dit is verblydend om daaraan te dink dat Amerikaanse fabrikante die probleem van die produksie van 'n entstof wat sowel veilig as doeltreffend is, op 'n bevredigende wyse opgelos het.

cines are biologically active only in the form of attenuated strains. Indeed, the view has been expressed that poliovirus vaccine is effective only in so far as a certain small number of live virus particles actually survive the process of inactivation.

Whatever the true scientific position may be, it is clear that there is no justification for abandoning the stringent test methods laid down and it is gratifying to know that the American manufacturers have solved the problem of producing a vaccine that is both safe and effective.

THE COXSACKIE VIRUS INFECTIONS

A BRIEF REVIEW

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The Coxsackie and poliomyelitis viruses have much in common. Both are small. The poliomyelitis virus is 30 mμ in diameter and the Coxsackie is almost the same size. Both are remarkably stable over a wide pH range, being able to survive at room temperature for a week at a pH 4.0 to 8.0. Both can be stored for many years in 50% glycerol-saline at 4°C. Both resist freezing at −70°C. and, unlike many other viruses, can be preserved at −20°C.

In both Coxsackie and poliomyelitis infections the virus can be isolated from the throat in the early stages of the infection, and from the faeces for several weeks after the onset of the illness. The stool is the common source for the isolation of these viruses. In cases of aseptic meningitis, the Coxsackie virus has been isolated from the cerebrospinal fluid. This is unlike poliomyelitis, in no case of which has the polio virus been demonstrated in the cerebrospinal fluid.

The Coxsackie virus, like the polio virus, spreads rapidly through the family and the family contacts, and may result in epidemics.

Coxsackie B infections, such as pleurodynia or meningitis, and paralytic poliomyelitis have been reported in the same year, but the peak incidence of the two has not been at the same time. Another common observation, in some areas, is that the years of high incidence of Coxsackie B infection are also the years of low incidence of poliomyelitis. It has been suggested that Coxsackie B infection may prevent or modify, presumably by interference, a superadded poliomyelitis virus infection of the infected host.

The name Coxsackie is well established. It is derived from a town on the Hudson River (not far from New York) where the virus was first isolated.

That Coxsackie infection is not a new disease is evident from the literature of the last century in which cases of epidemic pleurodynia have been described. However, the isolation of the virus by Dalldorf and Sickler is a recent event (1947), made possible by employing suckling mice, which are highly susceptible.

IMMUNOLOGICAL TYPES AND ISOLATION

To-day many viruses are being isolated from faeces, either by tissue culture techniques or the use of suckling mice. Most of these viruses are either of the poliomyelitis or Coxsackie group, and a few belong to the ECHO group.

The Coxsackie virus can be recognized by its pathogenicity for suckling mice; adult mice are relatively unaffected. On the basis of the lesions produced in mice, two large groups can be classified. Thus in group A the mice develop a flaccid paralysis. Examination reveals Zenker's hyaline degeneration of voluntary muscles followed by active repair. There is no involvement of brain or the fat pads between the scapulae.

Mice infected with group B strains develop tremors, spasms, weakness and encephalitis. A

unique lesion is congestion of the interscapular fat pads, followed by necrosis and calcification. Myositis is present, but focal, occurring in the psoas, masseter and tongue muscles. Internal organs may be involved, e.g. the heart and the liver.

In the past few years several serologically distinct strains have been isolated in each group. Thus, to date, there are 19 accepted strains in group A and 5 strains in group B. A new group A strain has recently been isolated in Italy by Archetti,[1] but it is not neutralized by any of the antisera of strains 1 to 19. It produced a relatively severe aseptic meningitis in various parts of Italy in 1955. In 53 cases Archetti isolated the virus directly from the cerebrospinal fluid.

Of interest is the behaviour of a Coxsackie A7 strain isolated in Russia from an admixture of the stools from 2 cases of fatal bulbar paralysis. The virus caused paralysis and the typical histological lesions of poliomyelitis after intramuscular or intracerebral inoculation of monkeys. It also caused paralysis and death in suckling mice. It was not neutralized by any of the 3 types of poliomyelitis antisera. The Russian workers therefore called it poliovirus type 4. However, Habel and Loomis[2] found this virus to be Coxsackie A7. They confirmed it was capable of producing polio-type lesions in monkeys and also that the American strain of A7 was likewise pathogenic for monkeys.

Growth in Tissue Culture. Several of the Coxsackie viruses can be grown in tissue culture. They produce a degenerative change in the cells (cytopathogenic effect) as do the polio viruses, but the incubation period is usually longer and the degenerative changes come about more slowly. All the types of group B virus can be isolated by monkey kidney tissue culture, but in group A only A9 can thus be isolated. However, group A strains have been isolated in human uterine tissue cultures and others in HeLa cells (a strain of tissue culture propagated from a squamous carcinoma of the cervix).

Growth in Mice. Mice aged 1–3 days old can be used, but in the case of some strains it is best to use mice not older than 24 hours. The stools of the patients are prepared as for the isolation of poliomyelitis viruses, and the material inoculated subcutaneously, intra-cerebrally or intraperitoneally into the mice.

EPIDEMIOLOGY

The Coxsackie viruses are world-wide in their distribution. Several types have been isolated in South Africa, both at the Poliomyelitis Research Institute, Rietfontein, and at the C.S.I.R. Virus Research laboratory of the University of Cape Town.

Source of the Virus. The viruses have been isolated from the pharyngeal secretions and the faeces of ill as well as apparently healthy persons. They have been isolated from the cerebrospinal fluid in cases of aseptic meningitis, and are present in high titre in the lesions of herpangina.

While the available evidence indicates that Man is the main source of spread of infection, the recovery of these viruses from flies and sewage suggests that infection may also spread indirectly by contamination of food and water.

Prevalence. According to Parrott,[3] a certain percentage of persons tested, particularly during summer, will be found to harbour group A viruses, regardless of their state of health. Thus the National Institutes of Health, Bethesda, found that from 1.5 to 7.5% of several groups of healthy or non-specifically ill persons tested during the summer were harbouring group A Coxsackie viruses, particularly those strains associated with herpangina. These findings are substantiated by investigations in other countries.

Season. The general experience is that the Coxsackie viruses are more prevalent in the summer and the autumn.

CLINICAL PICTURE

The incubation period varies from 2 to 5 days but may be 9 days or longer. The Coxsackie viruses, like the polio viruses, may produce an inapparent infection which results in immunity. On the other hand, unlike polio, severe infections carry a good prognosis (with the exception of myocarditis neonatorum) and permanent damage is not a feature.

Certain group A viruses may produce the picture of herpangina, and the same viruses may also produce a febrile illness with pharyngitis. These are A2, A5, A6, A8 and A10. Aseptic meningitis has been associated with A7 and A9 as well as with the group B viruses. The group B viruses are also responsible for epidemic pleurodynia or Bornholm disease and cases of myocarditis neonatorum.

It has been suggested that acute benign pericarditis is due to Coxsackie viruses. This may well be so, but it is still unproven.

Herpangina (*Vesicular Pharyngitis*). The disease usually occurs in young children in epidemic form in summer. It is uncommon in adults.

The onset of the illness is sudden, with fever varying from 101° to 105°F., and usually does not persist for longer than 2 to 3 days. Young children may have convulsions with vomiting, whilst older children may complain of headache, myalgic pains, dysphagia and sore throat. Within 48 hours lesions appear in the throat. These are minute papules or vesicles which progress to shallow, greyish ulcers surrounded by a zone of erythema. As a rule the lesions are few in number (2 to 5) but may be as many as 10 to 15. They are small, averaging 1–2 mm. in diameter, are situated on the anterior pillar of the tonsillar fauces and, less frequently, on the soft palate, the uvula and the tonsils. The tongue is uncommonly involved. These lesions persist for 1 to 5 days.

There is complete absence of cough or rhinitis and most patients do not appear acutely ill.

The illness lasts only a few days and immunity to the infecting strain appears permanent, but the presence of other strains capable of producing the symptoms may result in a repetition of the illness.

Febrile Pharyngitis. This is virtually herpangina without the characteristic pharyngeal lesions.

Siblings of patients with herpangina often have a similar illness, manifest by a diffuse pharyngitis and pyrexia but no ulcerative lesions. Most of them harbour the same type of virus as the contact with typical herpangina.

Epidemic Pleurodynia or Bornholm Disease. The clinical syndrome of epidemic myalgia was first observed in Iceland in 1856 by Finsen, who called it pleurodynia. Sylvest, a general practitioner in Denmark, recorded an epidemic of myositis on the island of Bornholm in 1930 and stressed the epidemicity and the intermittent muscular pains.

It has been proven that Coxsackie Group B viruses are the etiological agents, the commonest causal strain being B3.

The onset of the disease is sudden, with fever, headache and a spasmodic pain. There is usually an underlying ache with sudden, often excruciating, painful exacerbations lasting 5–10 minutes; hence the name, devil's grip. In some the pain is abdominal and has been mistaken for acute appendicitis or some other form of 'acute' abdomen. In others it is a chest pain varying in distribution, often widespread over one side of the chest and sometimes combined with abdominal pain. In a few a friction rub may be present, indicating an underlying pleurisy.

Most patients have some headache. Not infrequently it is severe and may show the same periodicity as the chest pains.

As a rule there is a pyrexia of 101° to 103°F. This may fluctuate quite considerably during the day. Pains in the limbs may be present, often typically spasmodic and at times associated with paraesthesia.

The average duration of the illness is 4-6 days in children, and longer in adults. However, relapses are frequent, occurring in about 30% of patients. These relapses can occur early, i.e. within a few days after apparent recovery from a short illness, or after 1–3 months. The relapse may be as severe as the original illness.

It is common to have several members of a household affected, after one member becomes ill.

Complications: Aseptic meningitis complicating pleurodynia has been described in several epidemics. A few patients develop orchitis, which may be bilateral but subsides in a few days.

Biopsy of affected muscles shows a myositis. The virus has been isolated from such lesions, but this procedure is *not* to be recommended.

Aseptic Meningitis. The Coxsackie viruses which have been isolated from cases of aseptic meningitis are A7 and A9, and B1 to B4, chiefly B2 and B3. According to Gear *et al.*,[4] Coxsackie Group B is the commonest cause of aseptic meningitis in Johannesburg. These workers, as well as others, have isolated the virus from the cerebrospinal fluid.

Most cases occur in children, who present with fever, nausea, vomiting, headache, drowsiness, pain in the neck and back and signs of meningitis.

The cerebrospinal fluid cell count is usually under 200 cells per c.cm. Lymphocytes usually predominate but polymorphs may be present in equal numbers. Protein values are not above 45 mg. per 100 ml. and the sugar and chlorides are normal.

The prognosis is good.

Myocarditis Neonatorum. In recent years a few cases have been described of acute myocarditis with cardiac failure in newborn infants. One such epidemic in Johannesburg was described by Javett *et al.*[5]

Like poliomyelitis, the onset of the illness may be sudden or it may be biphasic, the early or minor signs of slight pyrexia, and perhaps some looseness of the bowels, being followed by improvement and then by an exacerbation

after an interval of a few days. The baby then becomes acutely ill with pyrexia, cyanosis and a tachycardia out of all proportion to the elevation of the temperature. The illness may run an acute fulminating course terminating in circulatory collapse or in apparent complete recovery. The electrocardiogram shows evidence of myocardial damage and, in those infants who die, autopsy reveals a patchy, but intense, myocarditis.

On occasions the virus, usually Coxsackie B3, has been isolated from the myocardium.

The disease is a serious one and the mortality rate is high.

DIAGNOSIS

The diagnosis of Coxsackie infections is made by demonstrating the virus in the pharyngeal secretions, the faeces and the cerebrospinal fluid, and a rising antibody titre in the blood (shown by neutralization tests using paired acute and convalescent sera.)

TREATMENT

There is no specific treatment for the Coxsackie infections. Their management is therefore entirely palliative.

OPSOMMING

Die skrywer verstrek 'n oorsig van die eendersheid en die verskille tussen die Coxsackie- en die poliomiëlitis-virus, insluitende die afsondering en herkenning van die immunologiese tipes van eersgenoemde.

Nadat sy 'n beskrywing van die epidemiologie gegee het, teken sy 'n kliniese prentjie van die verskillende Coxsackie-virusinfeksies, en sluit af met enkele aantekeninge oor diagnose en behandeling.

REFERENCES

1. Archetti, I. (1957): *Quoted by* Dalldorf, G. *in* Ann. N.Y. Acad. Sci. (1957): **68,** 211.
2. Habel, K. and Loomis, N. Y. (1957): Proc. Soc. Exp. Biol Med., **95,** 597.
3. Parrott, R. H (1957): Ann. N.Y. Acad. Sci., **68,** 230.
4. Gear, J., Measroch, V. and Prinsloo, E. R. (1956): S. Afr. Med. J., **38,** 806.
5. Javett, S. N., Heymann, S., Mundel, B., Pepler, W. J., Lurie, H. I., Gear, J., Measroch, V. and Kirsch, Z. (1956): J. Pediat., **48,** 1.

STERNAL DEPRESSION

AS A CAUSE OF DIFFICULTIES IN DIAGNOSIS

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(*Concluded from p.* 198)

CASES COMPLICATED BY ORGANIC CARDIAC DISEASE

Patients in whom there is definite organic disease as well as pectus excavatus may be very difficult to assess when it becomes a question of the advisability of surgery.

Case 3: Mrs. S., a married woman of 41, had suffered from growing pains as a child. She was perfectly well and able to play good tennis until she was 36 years old. Since then she has suffered from increasing dyspnoea and 3 years ago symptoms of heart failure necessitated digitalis and Mersalyl. On one occasion this therapy was stopped and she became very oedematous with liver pain and orthopnoea. Her dyspnoea increased until she was unable to take the slightest exertion without extreme breathlessness. She had one attack of haemoptysis and developed orthopnoea just before she was referred for consultation.

She had a deep cup-shaped sternal depression (Figs. 16–18), but her chest moved well. The neck veins pulsated and were typical of tricuspid incompetence. Her apex beat was in the posterior axillary line, was forceful and left ventricular in type, but a right ventricular heave was also palpable. She had a grade IV–V apical mid-diastolic murmur and a II–III systolic thrill at the lower left sternal border. At the left sternal edge there was a grade V–VI rough, pan-systolic murmur. She suffered from moderate pitting oedema and her liver (6 inches below the costal margin) was grossly pulsating.

The cardio-thoracic ratio was 62% and the heart was completely displaced into the left hemithorax. The left atrium was enlarged. It was not possible to say which of the other chambers was enlarged. Kerley's lines were present.

The ECG tracings showed partial right bundle branch block, enlargement of the left atrium and a marked digitalis effect.

At rest her arterial oxygen was 93–95%. After 2 minutes' effort (10 x 20 cm. step) her

maximum drop was to 90% and on 100% oxygen her arterial oxygen rose to 100% in 20 seconds. Cardiac catheterization revealed marked elevation of pulmonary arterial and right atrial pressures. The pulmonary artery showed a systolic pressure of 90 mm. Hg, a diastolic pressure of 40 and a mean pressure of 63 mm. Hg. The right auricular pressures were 27.2 mm. and 14 mm. Hg diastolic, and indicated marked tricuspid incompetence.

Respiratory function tests showed some reduction in the vital capacity and in the maximum breathing capacity, but these results were relatively normal and in striking contrast to the patient's severe symptoms. This contrast indicated that the dyspnoea was primarily cardiac rather than pulmonary.

The problems presented by this patient were:

1. How much of her disability was due to mitral stenosis and what degree of incompetence and left ventricular failure was present?

2. How much, if any, of her disability was due to her sternal depression?

Even catheterization could not help on these points. There was a reasonable chance that significant mitral incompetence was present (*vide* absence of extreme right ventricular enlargement, borne out by the ECG examination, in the presence of a heart of this size and a normal first apical sound, absence of an opening snap and good function until 5 years before).

At the same time, the loud systolic murmur could be due to the sternal depression, of which it is a feature, and the dislocation of the heart could account for the absence of the opening snap.

Clinically the patient's symptoms and signs pointed to obstruction at the mitral valve and, if the case were due to a tight mitral stenosis, the tricuspid incompetence was not an important contra-indication to operation.

In view of her history (especially the onset of pulmonary oedema and haemoptysis), pointing to obstruction at the mitral valve as her main difficulty, it was decided to do a valvotomy, though opinion was very divided about its wisdom.

At operation (Mr. Lichter) a tight mitral stenosis was found and dilated.

She has had enormous benefit from this operation and now leads an active and vigorous life. The pulsations in her neck have dis-

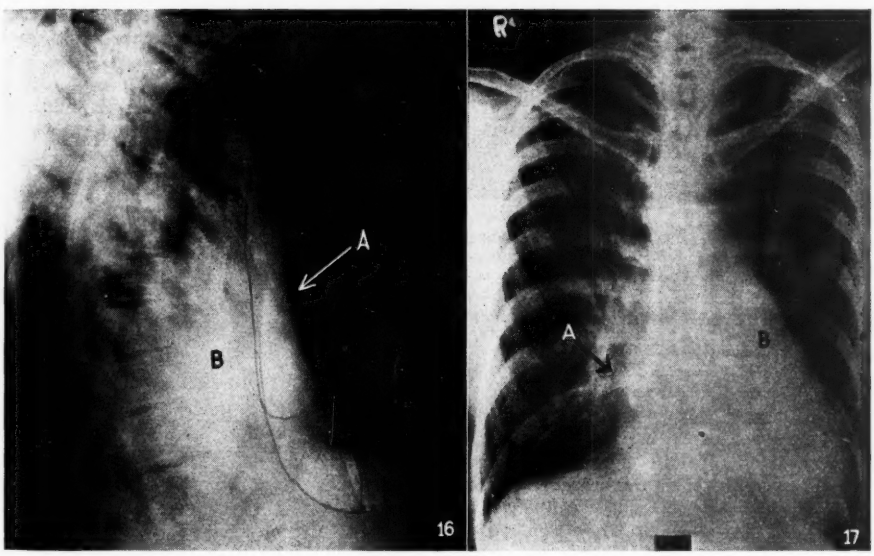

Fig. 16. Depression of the sternum and prominence of the right border of the heart.
A. Sternum
B. Heart

Fig. 17. Dislocation of the heart into the left thoracic space and prominence of the pulmonary shadows.
A. Pulmonary artery
B. Heart

appeared, as has her mid-diastolic murmur, but her harsh systolic murmur remains.

Two further patients with depressed sternums, who also suffered from mitral stenosis, have been seen. Both had systolic as well as diastolic murmurs and the question of mitral incompetence was entertained; but they presented no real difficulty once the sternal malformation was taken into consideration, and operation produced good results in both cases.

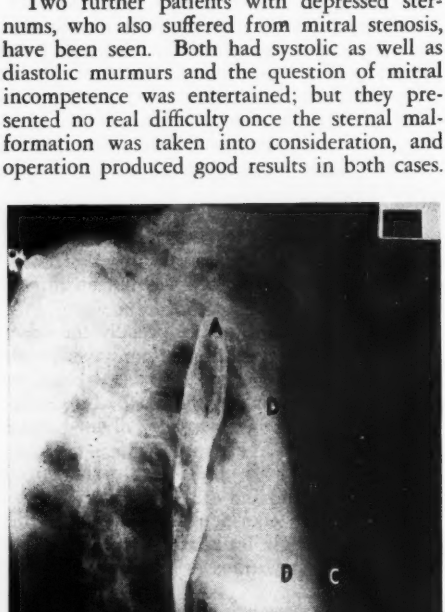

Fig. 18. Definite enlargement of the left auricle, but in no view is there any other chamber enlargement demonstrated although the cardio-thoracic ratio is increased.

A. Oesophagus
B. Left auricle
C. Breast
D. Sternum

DISCUSSION

This paper deals with difficulties in diagnosis attributable to the presence of sternal depression and therefore the question whether sternal depression can cause pulmonary or cardiac insufficiency *per se* is not dealt with.

If it does, it seems that it rarely occurs after puberty. Those who suffer from sternal depression are asthenic and easily fatigable types, prone to psychogenic disturbances such as hyperventilation. This must be borne in mind in assessing the clinical picture.

The systolic murmur (especially a late systolic murmur which, by mistiming the heart sounds, can easily be mistaken for a mid diastolic murmur), coupled with the X-ray appearances described, can easily lead to an erroneous diagnosis of mitral stenosis or incompetence.

Compression of the heart may distort the valves so that proper and complete closure is impossible and a degree of mitral or even tricuspid incompetence may well actually exist; but if the myocardium and the valves are healthy this (if it exists) is apparently well tolerated.

Evans[3] describes 2 cases of an aortic diastolic murmur for which no cause other than sternal depression could be found. No case of a diastolic murmur due to sternal depression was observed in this series. They must be rare, but when they do occur it must be a matter of great difficulty to differentiate organic aortic or pulmonary incompetence.

It is stressed that the minor degrees of the deformity may be overlooked and are more likely to lead to mistaken diagnosis of cardiac disease than gross degrees of sternal depression.

Occasional cases of sternal depression plus organic cardiac disease may be most difficult to assess and even catheterization and all modern techniques may not help our decision, which eventually must be based on a careful clinical assessment.

It may be impossible to be certain whether some degree of ventricular septal defect does or does not co-exist along with sternal depression, but at present this decision is only of academic value.

CONCLUSIONS

1. The signs and symptoms of 68 patients suffering from sternal depression are described and discussed.

2. Sternal depression may cause a mistaken diagnosis of mitral disease to be made.

3. Sternal depression occurring with rheumatic heart disease may render the functional assessment of the valvular lesion difficult, especially as regards the advisability of surgery.

4. A high proportion of patients with sternal depression (10.5%) also exhibit other congenital abnormalities.

5. Patients with sternal depression are usually thin and asthenic.

I am indebted to Mr. D. Adler for 2 cases quoted in this paper; also to Dr. Maurice McGregor for catheterization investigations.

I wish to thank Mr. Shevitz and the Photographic Department of the University of the Witwatersrand Medical School for the photographs.

OPSOMMING

1. Die tekens en simptome van 68 pasiënte met induiking van die borsbeen word beskryf en bespreek.

2. Induiking van die borsbeen kan tot gevolg hê dat 'n foutiewe diagnose van myterkwaal gedoen word.

3. Induiking van die borsbeen gepaard met rumatiekagtige hartkwaal kan die funksionele evaluasie van die klepletsel moeilik maak, veral wat betref die raadsaamheid al dan nie van chirurgie.

4. 'n Hoë persentasie van die pasiënte met induiking van die borsbeen (12.5%) vertoon ook ander aangebore abnormaliteite.

5. Pasiënte wat ly aan 'n induiking van die borsbeen is gewoonlik maer en kragteloos.

REFERENCES

1. Evans, W. (1946): Brit. Heart J., **8**, 162.
2. Evans, W. (1952): *Cardioscopy*, pp. 13–18. Butterworth & Co., Ltd. London.
3. Evans, W. (1954): Personal communication.
4. Smith, K. S. (1930): Middlesex Hosp. J., **30**, 43.
5. Teplick, G. and Drake, E. N. (1946): Amer. J. Roentgenol., **56**, 721.

CRANIOSTENOSIS, ASYMMETRY AND GROWTH OF THE SKULL

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(*Continued from p.* 148)

INTRACRANIAL PATHOLOGY: DEFICIENT GROWTH STRESSES

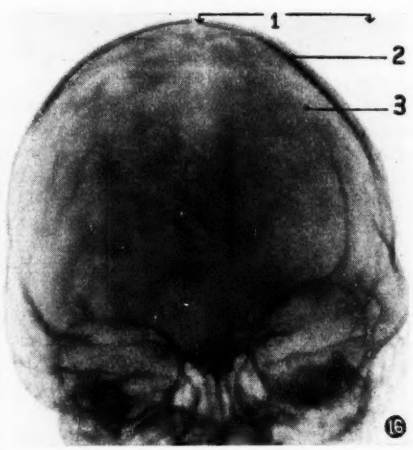
A CASE OF CEREBRAL HEMIATROPHY

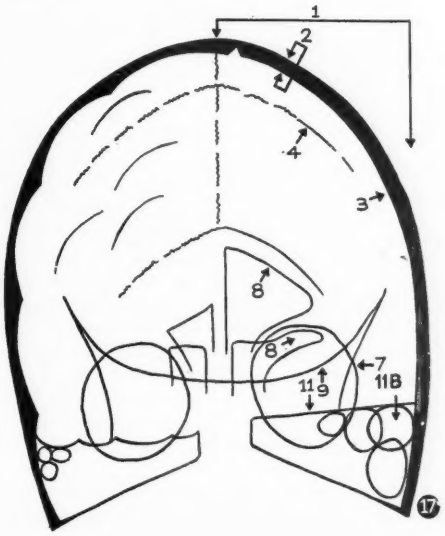
A child of 6 years was admitted for a right infantile spastic hemiplegia and was eventually subjected to hemispherectomy. The sphenoidal fissure view shows a small, thick, smooth left hemicranium with only a few convolutional impressions in the temporal region (1, 2, 3 in Fig. 16). The left orbit, lesser wing and petrous temporal are elevated. Pneumatization of the left petrous is excessive and the left frontal sinus, though poorly developed, is still larger than the right. The sutures on the left show early fusion. The groove for the superior sagittal sinus and the crista galli are displaced to the left.

Dyke, Davidoff and Masson[6] described the signs of unilateral cerebral atrophy as:

1. Thickening and smallness of one side of the vault (2 and 1 in Fig. 17).

2. Hypertrophy of the ipsilateral ethmoid, frontal and temporal air cells (8 and 11*b* in Fig. 17).





3. Elevation of the orbit and of the petrous bone on the same side.

To this description McRea[13] added:

4. Smoothness of the petrous ridge (11*a* in Fig. 17).

5. Displacement of the groove for the superior sagittal sinus (5 in Fig. 17).

6. Early closure of the sutures.

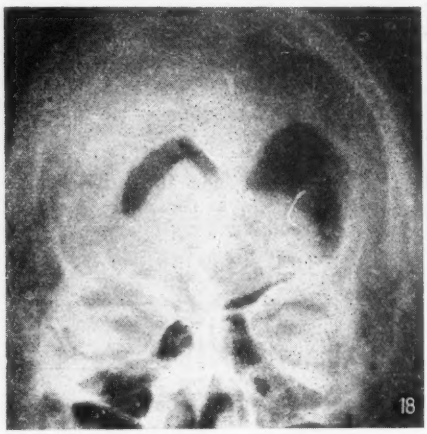
Thirty-four cases of known cerebral atrophy were examined to determine the incidence of these signs, without any useful result. All the signs occurred together only once, but in many cases a diagnosis could be made.

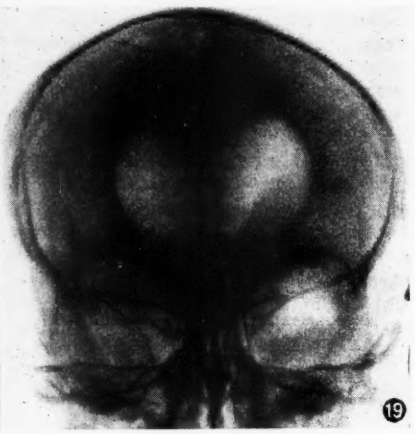
Thickening and smallness of one side of the vault was always accompanied by a loss of convolutional markings, and this was reliable evidence of cerebral atrophy. Elevation of the orbit and of the petrous bone were next most common. An unexpected finding was the variation in the position of the lesser wing of the sphenoid. In 8 cases with gross hemicranial atrophy the lesser wing was elevated in 3, depressed in 3 and symmetrical in 2, but in no case was there marked displacement where the skull manifested the changes of cerebral atrophy alone, i.e. without a porencephalic cyst.

Fig. 18 is a further example of cerebral atrophy. The left hemicranium is small because of elevation of the base, so that the orbit and the petrous temporal are high. The asymmetry of pneumatization of the frontal sinuses is within normal limits, but the very large left anterior ethmoidal and temporal air cells are more in keeping with cerebral atrophy. The left lateral ventricle is dilated and the ventricles are displaced to this side.

Fig. 19 is from a case of a mentally retarded child of 2 years. The left lesser wing is slightly elevated and the inner table on both sides is perhaps a little smoother than it should be for a child of this age. On these doubtful signs a diagnosis of cerebral atrophy was suggested, and it was confirmed by the dilated lateral ventricles on air encephalography. With more experience my enthusiasm has been tempered with caution, and I should to-day hesitate to say that there is anything abnormal about this skull *per se*.

Fig. 20 is from a patient with epilepsy with a haemangioma in the distribution of the left fifth nerve. It shows the characteristic double wavy line of calcification of Sturge-Weber's syndrome. The hemicranium is small but it is not thick or smooth, and the convolutional impressions on the inner table, though sparse, are of normal depth. Other views showed ipsilateral hypertrophied sinuses, elevation of the orbit and of the petrous bone, and elevation of the lesser wing. I have seen this curious combination of a small hemicranium without thickening or smoothness of the vault in other cases of Sturge-Weber syndrome, though I think it is commoner with these features.



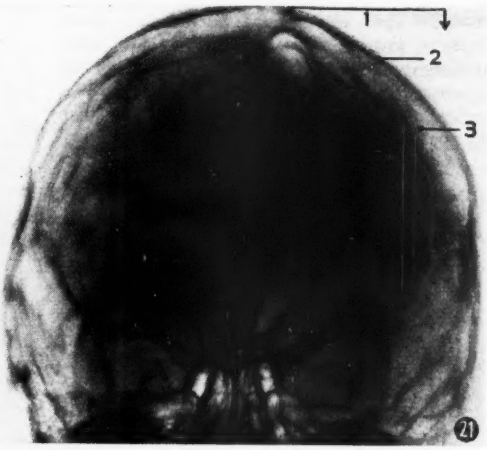


A CASE OF STURGE-WEBER SYNDROME WITHOUT CALCIFICATION

A girl aged 9 years was admitted for convulsions and a right spastic hemiplegia. She had a port-wine naevus in the distribution of the first and second divisions of the left trigeminal nerve.

The left hemicranium was small and the vault normally thick but with sparse, shallow convolutional impressions (1, 2 and 3 in Fig. 21). The lambdoid suture was narrower on the

left. The left frontal sinus was much larger but the ethmoids were symmetrical. The left orbit and the lesser wing were elevated. The linea innominata was normal. The left petrous temporal was slightly elevated, and normally indented and pneumatized. The air encephalogram showed a marked shift of the ventricles to the left, but there was little dilatation.



The small hemicranium, smooth inner table, large ipsilateral sinus and elevated petrous and orbit are signs of cerebral atrophy; but the vault was less thick and smooth than would be expected with such a small hemicranium in the ordinary types of cerebral atrophy. This inconsistency, i.e. a small hemicranium without proportionate thickening or smoothness seems to be not uncommon in the Sturge-Weber syndrome. It may be related to the vascularity associated with the naevus.

INTRACRANIAL PATHOLOGY: MIXED GROWTH STRESSES

A CASE OF A PORENCEPHALIC CYST

A mentally retarded boy aged 8 years was admitted for a right spastic hemiplegia from the age of 6 months. He had no fits. On the right side there was weakness of the sixth and seventh cranial nerves, spasticity of the limbs and an extensor plantar response. The cerebrospinal fluid contained 12 lymphocytes per c.mm. The rest of the examination was normal.

Fig. 22 shows a slightly enlarged left hemicranium with diminished convolutional markings and a thinned vault. The left orbit, the lesser wing and the petrous temporal are elevated. The linea innominata, sutures and pneumatization are normal. The air encephalogram shows a large porencephalic cyst (↑) with dilatation of the left lateral ventricle (⮝), but without displacement of the ventricular system.

McRea[13] stated:

'Occasionally they (the parietal and temporal squama) were thin on the small side, and in such cases an underlying collection of fluid in the brain or in the subarachnoid spaces could be postulated.'

Pendergrass and Perryman[17] suggested that this thinning of the vault was due to the transmission of the pulsating intraventricular pressure through the fluid in the cyst to the adjacent inner table.

These authors, and many others, regarded the cysts as atrophic or degenerative conditions, and they found difficulty in explaining the thinning of the vault that occurred instead of the thickening that usually accompanies the cerebral atrophies. The thinning of the skull

indicates that the cysts are the site of growth. That is not to say that they are the site of a measurably high hydrostatic pressure, for both the brain and the skull adapt to them, but their cystic shape and the reaction of the skull are evidence of growth. These cysts are associated with diffuse hemiatrophy of the cerebrum and consequent thickening of the vault, which is thin only where it is adjacent to a superficial growth. Thus both factors are present here. Excessive growth takes place in the cyst, with thinning of the adjacent overlying vault, but elsewhere the vault thickens secondary to the cerebral atrophy. This is clear evidence that the phenomena under discussion have nothing to do with the Pascal's law and the distribution of hydrostatic pressure within the cranium, but are simply the reaction of one tissue (the skull) to another (the brain). If the cyst is large enough the whole hemicranium may be enlarged and its vault thinned in spite of the complete replacement of the hemisphere by the cyst.

The degree of elevation of the lesser wing in this case is greater than that which occurred with cerebral atrophy without cysts, although it is not as marked as with other space-occuying lesions.

(*To be continued*)

A MEDICAL ATLAS

A CASE OF LEPROSY IN A NATIVE MALE

PRESENTING WITH MACULAR-ANAESTHETIC AND TUBERCULOID FEATURES

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It is not infrequent in dermatological or venereological practice to find non-European patients who complain of having 'funny marks on the genitals and all over the body'.

Such a patient was a Native male aged about 28 years, who presented himself recently at Baragwanath Hospital. Figs. 1 and 2 illustrate the dermatological condition of his genitals. The inexperienced practitioner, saddled with a knowledge of the high incidence of syphilis in the Bantu community, is more than likely to exclude leprosy from the differential diagnosis, especially when he finds that the skin lesion is associated with a positive Wassermann reaction. Errors of clinical judgment could be largely avoided if more attention were paid to

(*a*) The clinical appearances of the presenting skin condition; and

(*b*) The use of specific bacteriological investigative procedures.

(*a*) *The Clinical Appearances of the Skin Condition.* The genital skin lesions were infiltrated, with clear-cut, definite edges. They were mainly of the minor grade (minor tuberculoid), in that the infiltration was of slight degree. Close inspection of the edge of the lesion showed that it had a 'pebbled' character due to a series of papules around the periphery of a macule.

In parts the edges of the lesions were very well marked and even grossly infiltrated, suggesting a progression to the major tuberculoid type of leprosy. The superficial cutaneous nerves supplying the area were not found to be thickened. On testing the area of the skin lesions for sensory deficit, it was found that there was some loss of tactile, pinprick and pressure sensation. Clinically, then, the genital lesions were consistent with the minor tuberculoid type of leprosy.

The skin lesions, as they affected the back of the patient, were of the maculo-anaesthetic type. The macules were hypo-pigmented, with a clear-cut, erythematous edge. The surface had a dry appearance and the skin was rough. There was also a definite loss of tactile sensation and a degree of loss to heat sensibility. These clinical findings were consistent with those found in leprosy.

(*b*) *The Bacteriological Investigation of the Skin Condition.* Appreciation of the procedures utilized in the diagnosis of leprosy calls for a knowledge of the pathology of the condition. Leprosy is caused by the *M. leprae*. These organisms are introduced into the skin, and from the skin they enter into the area of the axon plasmic filaments of the nerves. They then multiply within the axis cylinder, gradually extend up the nerve, and subsequently

burst out of the nerve and pass to the deeper parts of the corium.

The character of the tissue reaction then determines the form of the disease. If the tissue response is adequate, the tuberculoid form of the disease results; in time the organisms become anchored in the skin by the formation of tubercles or epithelioid foci and are, as a result, prevented from spreading. This tuberculoid form of the disease is, accordingly, markedly self-healing.

If, however, the tissue response is poor or absent, then the lepromatous form of the disease develops. In this the organisms do not become anchored in the cutaneous tissues, but are left free to multiply in the macrophage cells, which then carry them to the deeper parts of the corium and, ultimately by way of the lymphatics, to the whole of the reticulo- endothelial system, viz. the spleen, the liver and the bone marrow. Lepromatous leprosy, in contrast to tuberculoid leprosy, shows large numbers of bacilli in the skin, but with no giant cells or epithelioid cell foci which are responsible for the tubercle formation; in addition, however, the mouth, the pharynx, the trachea and the *nose* become involved in the lepromatous process.[1]

It is obviously necessary, therefore, in any case of leprosy, (quite apart from the pathognomonic clinical evidence which may be adduced) to carry out a bacteriological examination:

i. Of a skin smear; and
ii. Of a nasal smear.

Both examinations were made in the case of the present patient. The skin smear was taken by the 'scraped' incision method described by Wade and Rodrigues.[3] The most indurated part of a lesion was chosen. The nasal smear was taken by the method described by Rogers.[2]

M. leprae was found by the first method only, thus clinching the diagnosis tentatively made on the clinical findings.

If the diagnosis had not been bacteriologically confirmed, it would have been necessary to differentiate the condition from a number of other skin conditions such as lupus vulgaris, sarcoid, syphilis, fungus infection, psoriasis, granuloma annulare, lupus erythematosis, etc.

OPSOMMING

In enige geval van melaatsheid is dit nodig (heeltemal afgesien van die patognomoniese kliniese bewyse wat aangevoer word) om 'n bakteriologiese ondersoek te doen:

i. Van 'n velsmeer; en
ii. Van 'n neussmeer.

Albei ondersoeke is in die geval van die onderhawige pasiënt uitgevoer. Die velsmeer is verkry deur middel van die ‚skraap'-insnydingsmetode soos deur Wade en Rodrigues[3] beskryf. Die mees verharde deel van 'n letsel is gekies. Die neussmeer is verkry volgens die metode wat deur Rogers[2] beskryf is.

M. leprae is alleen deur die eerste metode aangedui. Dit was dus 'n tentatiewe bevestiging van die diagnose wat aan die hand van kliniese bevindings gedoen is.

As die diagnose nie bakteriologies bevestig was nie, sou dit nodig gewees het om die toestand te

onderskei van 'n aantal ander velkwale soos lupus vulgaris, sarkoïed, sifilis, swaminfeskie, psoriase, granuloma annulare, lupus erythematosis, ens.

I wish to thank the Department of Medicine of the University of Witwatersrand, Johannesburg, for permission to use the photographs of this case. The patient was originally seen by Dr. M. Rose of Baragwanath Hospital, Johannesburg. I wish to thank him for permission to use his basic clinical data for the purpose of this article.

REFERENCES

1. Cochrane, R. G. (1957): In *Sequeira's Diseases of the Skin*, edited by J. T. Ingram and R. T. Brain, pp. 586–605. London: J. & A. Churchill Ltd.
2. Rogers, L. (1946): *Leprosy*, pp. 218–221. Bristol: John Wright and Sons Ltd.
3. Wade, H. W. and Rodrigues, J. N. (1939): Int. J. Leprosy, **7**, 327.

ACUTE POLIOMYELITIS*

A STUDY OF THE CLINICAL MANIFESTATIONS OF FIFTY CASES

SEEN AT THE CHILDREN'S HOSPITAL, JOHANNESBURG, DURING THE 1948 EPIDEMIC

WITH SPECIAL REFERENCE TO THE MANAGEMENT IN THE ACUTE PHASE

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(*Continued from p.* 174)

III. CLINICAL FEATURES: AGE, SEX, SYMPTOMS AND SIGNS OF 50 CASES OF ACUTE POLIOMYELITIS

CLINICAL FEATURES

AGE

Most cases occur in the first decade of life.[5, 29, 30]

In the present series (composed entirely of children) 44 of the total of 50 (88%) occurred in those under 10 years of age.

In the Johannesburg epidemic of 1918, 143 of 145 cases (99%) occurred in patients under the age of 15 years. In the epidemic of 1944–5, 144 of 174 cases (83%) occurred in the under-15 age group. In the 1948 epidemic, however, 453 of a total of 623 cases (72%) occurred in the under-15 age group.[5]

The decrease in the incidence of the disease in the under-5 age group with, however, little or no change in the incidence in the older age group, has been noted in certain American cities during the past 30 years. In the past 10 years the decrease has reached a point where children under 5 years are no more likely to acquire the disease than those aged 10–14 years. Both these groups were affected only about half as much as the 5–9 year old children.[6]

In the 1948 Johannesburg epidemic the incidence in the 10–14 age group was half that in the 5–9 group, and the incidence in the under-5 years age group approximately 1.5 times that in the 5–9 age group.[5]

It would therefore appear that the age incidence of the disease in Johannesburg and elsewhere has changed in that the older children (including young adults) have been affected more frequently, while the children under 5 years of age have not been affected in the same numbers as hitherto.

SEX

Very little has been written regarding the sex incidence of the disease. However, Grulee and Panos[29] found that of a total of 464 cases 278 (60%) occurred in males and 185 (40%) occurred in females. In the Southern Australian epidemic of 1947–8,[18] of a total of 103 cases 55 cases (53%) occurred in males and 48 cases (47%) occurred in females.

In this series of 50 cases 30 (60%) occurred in males and 20 (40%) occurred in females.

It would seem that the male may be slightly more susceptible to poliomyelitis than the female.

PRODROMAL SYMPTOMS

In this series prodromata occurred in 31 of the 50 cases (62%). In Grulee and Panos' series[29]

* The References will be published at the end of the concluding article in this series.

prodromata were recorded in 163 of a total of 464 cases (28%). Table 2 shows the symptoms in the two groups:

Peabody *et al.*[30] stated that the most constant feature of the prodromal period was fever. They also noted drowsiness in many cases, irritability in others, and occasionally spontaneous pain, more rarely muscular twitchings and convulsions. They pointed out that in the Hesse-Nassau epidemic of 1909 over 50% of the patients had prodromal symptoms involving the respiratory tract. These symptoms varied from a persistent cold in the head and occasional cough and sore throat, to bronchitis and even bronchopneumonia.

TABLE 2: PRODROMAL SYMPTOMS IN POLIOMYELITIS

Symptom	*This Series of* 50 *Cases* (30 *Cases with Prodromata*)	*Grulee and Panos* (1948) 464 *Cases* (163 *Cases with Prodromata*)
Fever	25	Majority
Malaise	9	Majority
Headache	7	Majority
Sore throat	7	47
Vomiting	4	26
Irritability	2	Not specified
Generalized pain	3	Not specified
Abdominal pain	2	Not specified
Coryza	2	18
Convulsions	1	Not specified
Cough	1	Not specified
Constipation	1	Not specified
Rigor	1	Not specified
Rash	1	Not specified

In the Westphalian epidemic described by Krause[31] over two thirds of the cases had diarrhoea in the prodromal period.

In the 1907 New York epidemic[30] gastric symptoms such as anorexia, nausea and vomiting were observed in a large percentage of the cases.

In this series fever occurred in the great majority of cases and the most common groups of symptoms were fever and sore throat; fever, malaise and headache; or fever and headache. Vomiting, generalized pain, abdominal pain and coryza occurred in a small proportion of cases, and convulsions, cough, constipation, rigors and rash in only one case each.

Fever, malaise and headache occurred in most cases described by Grulee and Panos,[29] whereas sore throat, vomiting and coryza were much less frequent. They did not comment, however, on any of the other symptoms described in this series.

The interval between the end of the first phase of the disease and the commencement of the invasive phase ranged from 0.5–6 days with an average of 2 days in the diphasic pattern of the disease. In the straggling form, however, the symptoms were present from 1–7 days with an average of 4 days before the onset of the invasive phase. The overall interval in this series was 3 days, in Grulee and Panos' series[29] 4 days, while, according to Peabody *et al.*,[30] the average was from 1–3 days. The prodromal symptoms are, on the whole, non-specific and appear at the onset of almost any acute illness in childhood. The significance of the clinical pattern in the prognosis of the disease will be discussed later.

An analysis of the prodromal findings in epidemics during the past 40 years shows that comparatively little change has taken place in this aspect of the disease.

SYMPTOMS

The history of an illness is all important in attempting to define its symptomatology. Since this series was confined to children, the information was necessarily gathered from the mother in most instances and from the child, if old enough. During an epidemic most parents are extremely anxious at the onset of any trivial disease; consequently their observations reflect this anxiety. In many instances the parents were questioned several times in an attempt to gather more accurate data. Furthermore, much depended upon the intelligence of the parent and the time she chose to spend with and observe the child. In this series most of the children belonged to fairly intelligent and observant parents. Despite the obvious difficulties involved, it was nevertheless decided to compare epidemics in different countries with the epidemic in Johannesburg.

In most reported epidemics symptoms and signs are necessarily grouped together, but an attempt will be made to compare the symptoms before hospitalization in 4 epidemics with those in this series. The symptoms are summarized in Table 3.

Fever, together with pain in the neck, back and limbs was the commonest symptom in all the epidemics. However, muscle paralysis is not recorded as a symptom in most series and will be discussed in greater detail in the section dealing with signs.

In this series 16 cases (32%) presented with paralysis, noted by the mother in the period before hospitalization. In most of the epidemics used for comparison with this series

TABLE 3: SYMPTOMS IN THIS SERIES COMPARED WITH THOSE OF 4 SEPARATE EPIDEMICS

Symptoms	*Rockeller Monograph*[30] *and Draper's Monograph*[19] (115 *Cases*) (Peabody *et al.*, 1902)	*Malta Epidemic* 1942—1943[32] (464 *Cases*)	*South Australia Epidemic*[18] 132 (*Cases*)	*Minnesota Epidemic*[29] (464 *Cases*)	*Johannesburg Epidemic* 1947—1948 (50 *Cases*)
Fever	Probably always present	245 (53%)	126 (96%)	430 (93%)	35 (70%)
Stiffness of Neck or Back	64 (55%)	Not recorded	101 of 128 (79%)	Not recorded	20 (40%)
Muscle Praralysis	Only analysed in paralytic cases	Not analysed as a symptom	Not recorded	Not recorded	16 (32%)
Headache	28 (24%)	72 (15%)	106 of 124 (86%)	45 (53%)	15 (30%)
Neck, Back and Limb Pain	37 (32%)	275 (59%)	82 (73%) (113 cases investigated)	Not recorded	10 (20%)
Vomiting	25 (22%)	117 (25%)	84 of 113 (63%)	205 (44%)	7 (14%)
Retention of Urine	5 (4%)	Several cases—number not recorded	Not recorded	40 (9%)	4 (8%)
Convulsions	2 (2%)	13 (3%)	Not recorded	Not recorded	4 (8%)
Sore-throat	Not recorded	6 (1%)	45 of 108 (42%)	94 (20%)	3 (6%)
Abdominal Pain	Not recorded	42 (9%)	29 of 83 (35%)	45 (10%)	3 (6%)

paralysis was not recorded as a symptom, since many of the patient's entered hospital before the appearance of muscle weakness.

Headache was present in 15–85% of cases reported. This variation was probably due to the age of the patients reported in the various epidemics. In this series it occurred in 15 cases (30%). Older children and adults would complain of headache more frequently than younger children. There was nothing specific about the headache and it was distinct from the pain associated with stiffness of the neck. It usually lasted 24–48 hours and was often relieved by the lumbar withdrawal of 5 c.c. of cerebrospinal fluid. The headache had an aching quality, primarily located in the frontal region.

Pain, a prominent feature in most of the epidemics, was complained of in 10 cases (20%) in this series. In some instances it was associated with stiffness, in others it appeared in spasms or was induced by movement.

Stiffness of the neck or back or the two together was encountered frequently (16–79% of patients). In this series it was noted in 20 cases (40%). The reason for this discrepancy was probably due to the difficulty in eliciting a subjective complaint in a very young, irritable, apprehensive child. The patients actually complained of stiffness which prevented normal movement of the whole spine. Pain was frequently induced by movement of a stiff muscle or muscle group.

Vomiting was present in 14–63% of cases and occurred in 7 (14%) in this group. It occurred once or several times and, on occasion, was projectile. It bore no relation to the height of the intracranial pressure.

Abdominal pain was present in 6–35% of the cases recorded. Three cases (6%) in this series had abdominal pain. The pain was usually mild and generalized, but in some instances became severe enough to simulate an acute surgical abdomen. It usually persisted for 24–72 hours. Earlier observers,[19, 30] however, did not mention this symptom.

Drowsiness was present in 2 cases (4%) in the series. The mothers noted that the children tended to sleep all the time, but became restless and irritable when roused. Drowsiness was stressed as a common symptom by some authors[19, 30] and was present in 34% of the cases in the Malta epidemic,[32] but was not mentioned in the South Australia[18] or Minnesota[29] epidemics.

Difficulty in urination often associated with retention was present in 4 cases (8%) of this series. This symptom usually persisted for 24–48 hours and occasionally needed catheterization. In some instances it was present for shorter periods while in others it lasted for

several days and required some form of continuous drainage.

Generalized convulsions were noted in 4 cases (8%) in this series and in 2–80% in the other epidemics. Patients rarely had more than 2 seizures. On the other hand, muscle twitching was recorded in 10% of the earlier epidemics.[19, 33]

Diarrhoea was present in one case (2%) in this group. It was recorded in 1–6% of the cases in the other epidemics. The diarrhoea was usually mild and disappeared in 48 hours.

Constipation was complained of in one case (2%) in this series, but was mentioned in 2–16% of the cases in the various epidemics. On the other hand, it was present in most cases during the subsequent course of the disease.

Staggering was complained of in only one case (2%) in this series and was followed by a convulsion soon afterwards. It could therefore have been a prodromal manifestation. Staggering unassociated with any weakness or stiffness of muscles was recorded in from 2–5% of cases in some series. However, it was not mentioned in the 3 large epidemics analysed in Table 2.

Rigors and generalized sweating were each present in one case (2%). Rigors were noted in only 2% and 4% respectively of cases recorded in 2 epidemics. They were identical with rigors in other diseases. Generalized sweating out of all proportion to the height of the fever was recorded in only 2% of cases. Some authors[30, 33] stressed the frequency of this symptom in the earlier part of this century. In some patients the bed clothes were drenched. Miscellaneous symptoms such as loss of speech, cough, vertigo, delirium, oedema of the eyelids and transient joint swellings mentioned in other epidemics[18, 32] were not seen in this group of cases. Malaise, a frequent symptom of any disease in childhood, accompanied all the cases in this series, and was present in most cases described in other epidemics.

Sore throat, noted in 1–42% of the other series, occurred in 3 cases (6%) of this series. It was usually mild, persisting for 24–48 hours.

Abnormal gait was complained of in 3 cases (6%) in this series, the mother stating that the children walked stiffly. Examination confirmed their observations. The difficulty was due to stiffness of the sacrospinalis, hip and hamstring musculature. This symptom was not recorded in any of the other epidemics.

An analysis of the data of both earlier and more recent epidemics shows that the symptomatology of the disease has hardly changed. One of the notable differences between the epidemics in the earlier part of the century and the one occurring in Malta in 1942,[32] as compared with the 3 most recent epidemics, is in the incidence of drowsiness. The latter was a prominent feature of the former epidemics and an uncommon finding in the more recent outbreaks of poliomyelitis. The explanation for this ostensible change is probably related to the degree and type of encephalitic involvement.

Certain symptoms such as fever, headache and vomiting are common to both the prodromal and invasive phases of the disease. It is therefore very difficult to differentiate these phases when these symptoms alone are present. The presence of generalized body pain and stiffness of the neck and back are indicative of the disease itself.

SIGNS

A feature common to all cases described in this series was the excellent physical and mental status of the children. They were usually near the top of their class at school and frequently the most handsome, robust and intelligent children in the family. Wickman[4] commented upon the fact that the disease attacked 'robust, active, well fed and handsome children'. Thelander and Pryor[34] stated that the delicate, thin, handicapped child was not as frequently attacked as the sturdy child. They could find no evidence of any endocrine anomalies in some of their 'choicest and handsomest' children. Draper and Dupertius[35] noted that certain morphological features were more common in patients with poliomyelitis than in normal controls. Their work has been confirmed by Aycock.[36]

The most striking facial expression in 30 cases of this series was that of apprehension and anxiety. The eyelids were slightly retracted giving rise to a mild degree of exophthalmos and the frightened, suspicious facial expression was characteristic. In addition 12 of the cases had flushed cheeks together with circumoral pallor. The child was extremely irritable and cried out or whined when anybody approached the bed or attempted the slightest movement of any part of the body.

The combination of these features was in itself suggestive of a diagnosis of poliomyelitis in this epidemic. In addition, the eyes in some cases continuously moved around in all directions, whereas in others, movement of the alae nasi without accompanying respiratory embarrassment was obvious. This picture was

markedly different from that described in earlier epidemics.[30, 33] Peabody and Draper[30] commented upon the drowsy and even stuporose condition of their patients, who became very irritable when aroused, but soon relapsed into their drowsy state. Drowsiness was a feature in the 1942–3 Malta epidemic[32] but was not commonly seen in the other epidemics. On the other hand, Draper[19] observed several cases with a glassy, frightened stare. These presumably were similar to the apprehensive facies described (*vide supra*). Apprehension and anxiety appeared in the encephalitic and anoxic varieties of poliomyelitis in several of the more recent epidemics.[20, 29, 37]

Tachycardia was present in 44 cases. The duration of the tachycardia was identical with that of the temperature. It persisted from 1–10 days with an average of 4 days. A sudden increase in a relatively stable pulse rate or one tending to return to normal, occurred in 11 cases, 10 of which showed progression of the disease with attendant paralysis. The pulse rate was very rapid and irregular, due to ventricular extrasystoles, in 2 of the fatal cases.

Pyrexia was present in 42 cases, the temperature ranging from 99–105° F, rising to 106° F. preterminally in one case. The average temperature on admission was 102–103° F. and the fever usually persisted for 1–10 days with an average duration of 4 days. The author saw several cases in this epidemic in which the pyrexia persisted for longer periods—42 days in one case. Defervescence took place by lysis rather than crisis. As long as the temperature remained elevated in the acute phase of the disease, progressive weakness of involved muscles or the appearance of paralysis in other groups of muscles could take place. Furthermore, a sudden rise in either a sustained fever or one that was beginning to decline, usually denoted extension of the disease. This exacerbation was noted in 11 cases in this series, and in 10 the disease progressed. In only one case was paralysis noted 48 hours after the temperature had returned to normal. These observations were well described by Peabody *et al.*[30] They reported pyrexia averaging 2–4 days in most of their cases, up to 10 days in some and 2–3 weeks in exceptional cases. In the Malta epidemic,[32] however, the pyrexia averaged 2 days with most cases having fever for one day only.[33]

Muscle stiffness or spasm, an important sign, was usually present in the neck, spinal and hamstring musculature. It is commonly seen in meningeal irritation due to a variety of causes apart from poliomyelitis. It differed in poliomyelitis in that it could be present in other muscle groups apart from those mentioned and its duration often exceeded the inflammatory period affecting the meninges. In some cases it was associated with actual muscle pain, in others with stiffness of the affected muscle groups.

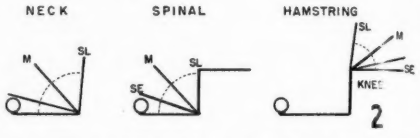


Fig. 2. Three grades of neck, spinal and hamstring stiffness SL=slight; M=medium; SE=severe;

The stiffness or spasm is best elicited by the clinical manoeuvres described in the section devoted to this particular phenomenon. It was divided into 3 varieties (Fig. 2). The mild form existed when stiffness could only be demonstrated on maximum flexion of the neck and spine and maximum extension of the knees, whereas the severe variety manifested pain upon the slightest attempt at movement. The moderate variety was present when motion through approximately half the normal range induced discomfort. Apart from the pain or discomfort induced at the particular site, the muscles could often be palpated as tender, firm structures.

Muscle stiffness of varying intensity was present in 41 cases, 12 of which showed slight stiffness, 9 medium and 20 severe stiffness.

TABLE 4: SEVERITY OF MUSCLE STIFFNESS IN 50 CASES OF ACTUE POLIOMYELITIS

Type of Poliomyelitis	*Muscle Stiffness*			
	Nil	*Slight*	*Medium*	*Severe*
Non-paralytic (17 cases) ..	0	8	5	4
Spinal paralytic (19 cases) ..	4	0	3	12
Bulbar paralytic (8 cases) ..	4	2	0	2
Bulbo-spinal paralytic (6 cases) ..	1	2	1	2
Total: 50 *Cases* ..	9	12	9	20

The incidence of the severe grade was much higher in the spinal paralytic group (12 of 19 cases), but was also present in 4 of the 17 cases of the non-paralytic variety. In all the cases presenting muscle stiffness, the neck,

spinal and hamstring muscles were involved, but in 3 patients this phenomenon was present in the pectoral, calf and hip muscles respectively as well. The duration of this symptom varied from 1–166 days. In the slightly stiff group the average was 4 days, in the moderate 12 days and in the severe 23 days.

The presence of muscle stiffness and its accompanying pain and discomfort was frequently observed in the cases seen by Wickman.[4] In the 1907 New York epidemic these features persisted for 1–6 days in 94 cases, 7–14 days in 178 cases, 14–28 days in 79 cases and 28–84 days in 35 cases.[33] In 3 cases, however, they lasted for several months. These figures are similar to those already described.

Paralysis. Lower motor neurone paresis or paralysis of one or several groups of muscles was present in 33 cases. Muscle weakness was graded as follows (Medical Research Council):[38]

0 = No contraction.
1 = Flicker or trace of contraction.
2 = Active movement with gravity eliminated.
3 = Active movement against gravity.
4 = Active movement against gravity and resistance.
5 = Normal power.

According to this classification, paralysis of muscle groups rather than individual muscles was as follows (Table 5):

TABLE 5: INCIDENCE AND SEVERITY OF MUSCLE PARALYSIS IN 50 CASES OF ACUTE POLIOMYELITIS

Muscle Groups	*Grade*				
	0	1	2	3	4
Face	7	0	0	0	0
Eye	1	0	0	0	0
Pharynx	1	0	0	0	0
Both upper limbs	3	0	1	0	0
Shoulder	3	0	2	2	0
Elbow	1	0	2	2	0
Wrist and fingers	0	0	1	2	0
Entire lower limb	3	0	3	1	0
Both lower limbs	5	0	1	1	0
Hip	0	0	2	0	0
Knee	0	0	4	1	0
Ankle and foot	1	0	3	1	0
Diaphragms and intercostal	2	0	1	1	1
Abdomen	0	0	2	0	0
Spine	0	0	0	0	3

It is interesting to note that in cranial nerve involvement all the affected muscles were completely paralysed (category 0) whereas the limb and trunk musculature showed 18 instances in the 0 category, nil in the 1 category, 21 in the 2 category, 11 in the 3 category and 4 in the 4 category. Hence most of the cases of palsied peripheral musculature fell into the second and third grades, but a substantial number were completely paralysed.

These findings illustrate the high incidence of bulbar involvement together with the frequent involvement of the extremities, especially the lower ones. The high incidence of diaphragmatic and intercostal paralysis, as well as quadriplegia, was due to the fact that these features were present in 2 of the 3 fatal cases. Spinal and abdominal musculature were infrequently involved.

In the upper limbs the abductors of the shoulder, especially the deltoid muscles, were most frequently affected; whereas in the lower limbs the extensors of the foot and knee were most frequently involved.

The main difference between the older and more recent epidemics has been in the increase in incidence of the bulbar variety of the disease, especially so in the 1946 Minnesota epidemic.[29] Furthermore, there has been a marked decrease in the number of cases with involvement of the abdominal and spinal musculature. The greater incidence of lower limb involvement remains the same, the distribution of the paralysis in limbs remains unchanged, and the abductor muscles of the shoulder, the flexors of the hip and the extensors of the knee and ankle remain the most commonly affected muscles in all epidemics. Of the cranial nuclei, those affected in order of frequency were the seventh, ninth and tenth, third and sixth, eleventh and twelfth and fifth nerves respectively, but the incidence varied in each epidemic.

Pain. There were several varieties of this important symptom.

i. The *spontaneous type* frequently mentioned by the older children and adults simulated the aching muscles of influenza in some cases, while others cried out with spasmodic severe knife-like shooting pains commencing in the spine and radiating down the legs and even arms. It simulated the stabbing pain associated with peripheral neuritis. Meningeal pain, also stabbing in nature, was present at rest, but was usually brought out by passive or active movements. It was usually localized to the neck and spine.

ii. *Induced pain* was commonest and usually associated with neck, spinal and hamstring muscle stiffness. Movements stretching the stiff muscles or squeezing them caused the patients a great deal of discomfort.

iii. *Cutaneous hyperaesthesia* mainly confined to the limbs was a symptom in some

cases. These patients resented the slightest stroking of the affected skin and even found contact with the bed clothes uncomfortable.

Pain was present as a symptom in 10 cases in this series. The distribution and subsequent paralysis are shown in Table 6:

TABLE 6: RELATIONSHIP BETWEEN THE SITE OF PAIN AND THE MUSCLE SUBSEQUENTLY PARALYSED

Type of Disease	*Site of Pain*	*Subsequent Paralysis*
Non-paralytic	Lower limbs	Nil
	Right shoulder	Nil
Spinal paralytic	All 4 limbs	Left leg
	All 4 limbs	Arms and legs
	Right leg	Right leg
	Neck, back and legs	Right leg
	All 4 limbs	Arms and legs
	Back and lower limbs	Left leg
	Back, left leg and left arm	Left leg and movements at both elbow joints
Bulbo-spinal	Left leg and abdomen	Pharyngeal muscles, right shoulder and right diaphragm

The frequency of limb pain in the spinal paralytic group is of some importance and its significance will be discussed in the section on prognosis. The presence of pain does not necessarily indicate paralysis of affected muscles and its complete absence in the cranial musculature which subsequently became paralysed was striking. As a sign of poliomyelitis, pain of one form or another was present in all cases exhibiting spinal and hamstring stiffness. In most of the writings in the literature pain was noted as a common feature of the disease at one stage or another, but no attempt was made to separate the spontaneous and induced varieties. Pains in the limbs, neck and back were noted in 469 cases (78%) of the series reported by Ruhräh and Mayer.[33] In the Malta epidemic[32] 275 cases (59%) exhibited pain in one form or another, and in the South Australia epidemic[18] 82 cases (73%) showed this symptom. It is therefore evident that pain was a frequent and important symptom and sign in both the older and more recent epidemics of poliomyelitis.

Reflexes. The routine reflexes elicited in every case in this series included: biceps, supinator, triceps, upper and lower abdominal, knee, ankle and plantar responses. Since the normal range of reflex activity is very wide, hyper-reflexia was noted when the slightest tendon stretch persistently resulted in a very brisk response. In some doubtful cases the intensity of the reflex response was judged in retrospect by comparing it with the normal reflex activity obtained at a later date. With these criteria, increased reflex activity or hyper-reflexia was demonstrated in 29 cases. In the spinal paralytic group several instances of hypo-reflexia in paralytic limbs were seen together with hyper-reflexia in non-affected limbs. The distribution of reflex change was as follows (Table 7):

TABLE 7: SUPERFICIAL AND DEEP TENDON REFLEXES

Reflexes	*Non-Paralytic*	*Spinal*	*Bulbo-Spinal*	*Total*
Hyper-reflexia	13	12	1	26
Normal	3	7	2	12
Decreased or Absent	1	11	4	16

Hyper-reflexia was present in 26 cases, hypo-reflexia in 16 cases, and normal reflexes in 12 cases.

Although hyper-reflexia was present in almost the same number of cases belonging to the spinal paralytic and non-paralytic groups, hypo-reflexia was much commoner in the spinal paralytic group. Nevertheless, one case in the non-paralytic group showed decreased reflexes, whereas the cases belonging to the bulbar variety demonstrated normal or increased reflexes. An extensor plantar response was not elicited in any case in this series, but in the 500 cases analysed by the author in this epidemic, this sign was noted in only 6 instances. The most frequent abnormality in reflex activity was that of hyper-reflexia which persisted for a period of from 1–6 days and usually disappeared as the acute phase of the disease passed over. Ruhräh and Mayer[33] noted absent reflexes in complete paralysis and decreased reflexes in partial paralysis. They stated that reflexes were only increased in the true cerebral spastic type of polioencephalitis. Peabody *et al.*[30] did not comment on the reflexes. Draper[19] and Horstman[20] described hyper-reflexia in the early stages of the disease, returning to normal reflex activity or changing to hypo-reflexia and even complete disappearance of reflexes, depending upon the progress of the disease. Brown and Baker[37] considered hyper-reflexia to be part of the encephalitic manifestations so commonly seen in all varieties of poliomyelitis.

Apprehension and irritability were present in 24 cases, 16 of which belonged to the paralytic group and 8 to the non-paralytic group.

In 5 cases belonging to the bulbar and bulbo-spinal groups, apprehension alone was present. The frequency of both these symptoms, especially the apprehension, has been stressed recently as part of the encephalitic manifestation of poliomyelitis.[37] This symptom has been noted by several authors.[20, 30]

Tremor of the outstretched hands was noted in 25 cases, 13 of which belonged to the spinal paralytic group, 6 to the non-paralytic and 3 to the bulbar and bulbo-spinal groups respectively. Marked tremor was present in 2 of the fatal cases who showed extreme apprehension as well. The 3 signs (apprehension, hyper-reflexia and tremor) were usually present together and lasted for the same period, viz. 1–6 days.

Constipation was a serious problem in 38 cases in this series. As no patient was allowed to remain constipated for longer than 4–5 days, it was not possible to determine the exact duration of this abnormality. Of the 38 cases, 16 belonged to the spinal paralytic group, 14 to the non-paralytic, 5 to the bulbar and 3 to the bulbo-spinal group. The symptom persisted for 1–3 weeks, after which time the children resumed their normal bowel routine. Peabody *et al.*[30] stressed the frequency of constipation in their series. Grulee and Panos[29] reported constipation in 257 (55%) of 464 cases, while 128 (30%) of 426 cases showed this symptom in the Malta epidemic.[32] On the other hand, diarrhoea was noted in only 8 instances (2%) of the 426 cases in the latter epidemic.

(To be continued)

NOTES AND NEWS : BERIGTE

NUTRITION SOCIETY OF GREAT BRITAIN

The Nutrition Society will hold a symposium on *Nutrition and Teeth* at the London Hospital Medical School, Turner Street, London, E.1, on Saturday, 4 October, 1958.

* * *

U.S. PHARMACOPEIA (U.S.P. XVI)

The next edition of the U.S. Pharmacopeia is scheduled for publication early in 1960. Three recently developed products have been selected for inclusion in this revision of the Pharmacopeia, viz. Chlorothiazide, Mecamylamine and Novobiocin.

* * *

Dr. S. Etzine, of Johannesburg, is overseas, where he is attending ophthalmic clinics in Rome and Lausanne.

Dr. Etzine is accompanied by his wife. They will be back in Johannesburg at the end of May.

* * *

THE WESTDENE PRODUCTS SCHOLARSHIPS FOR 1958

These scholarships have been awarded to the following medical students:

Cape Town University:
B. M. Kennelly .. .. 6th year
D. J. Clain .. .. .. 5th year
A. D. Ginsburg .. .. 4th year

Natal University:
I. M. Seedat .. .. .. 6th year
B. R. Maku and Miss S. Reddy 5th year
Miss R. Pather and D. R. de Wahl Matlhoke .. .. 4th year

Pretoria University:
G. J. Pistorius .. .. .. 6th year
R. E. van Schalkwyk .. .. 5th year
A. van Dam .. .. .. 4th year

Witwatersrand University:
G. Kos .. .. 6th year
Miss D. Hack .. .. .. 5th year
A. H. Rubenstein .. .. 4th year

E. & S. LIVINGSTONE LIMITED

MR. CHARLES MACMILLAN'S VISIT TO SOUTH AFRICA

Mr. Charles Macmillan, Managing Director of the well-known firm of publishers (E. & S. Livingstone Limited), is on a short visit to South Africa. Medical practitioners interested in meeting him for the purpose of discussing medical publications, may get into touch with him at the following addresses:

Johannesburg (Langham Hotel) 3 to 6 May.
Durban (Marine Hotel) 7 to 12 May.
Cape Town (Queens Hotel, Sea Point) 17 to 23 May.

* * *

Dr. B. W. Levinson, of Tara Hospital, Johannesburg, has been made a member of the British Society of Medical Hypnotists.

* * *

HOW BIG WAS YOUR TUMOUR?

NUTS, EGGS AND CANCER

The following list indicates estimated tumor sizes as recorded in hospital charts of the Indianapolis General Hospital and the Indiana University Hospitals. They were not made by students, technicians, or nurses. They were made by doctors, both resident and attending.

Egg, small egg, duck egg, goose egg, guinea egg, hen's egg, large egg, robin's egg, pigeon egg, quail egg, small pullet's egg, banty egg.

Walnut, hazelnut, hickory nut, English walnut, hulled walnut, hull of walnut, pecan acorn, unhulled walnut, large pecan, half a hickory nut, chestnut, large peanut, almond.

Grain of corn, few grains of maize, bean, kidney bean, navy bean, pea, lentil seed, soup bean, spilt pea or grain of wheat, millet-sized.

Orange, small orange, lemon, small lemon, lime, grapefruit, small grapefruit, large grapefruit, half the

size of a lemon, cherry, small plum, large orange, two large oranges, tangerine (large), half grape, melon, half a banana, dried prune, watermelon seed, peach.

Stuffed olive; cup cake—flat on top.

Dollar, dime, nickel, quarter, half a dollar, dollar and a half, dollar bill, mass the size of five quarters stacked on one another, a fifty-cent piece.

Baseball, third of a baseball, tennis ball, golf ball, billiard ball, ping pong ball, football-sized mass, volley ball.

Fist, hand, thumb, tip of thumb, end of little finger, child's fist, as big as a man's head, baby's head, as big as two thumbs.

Saucer, dinner plate, tea cup, coffee cup.

Pencil point, pin head, eraser in a lead pencil, matchhead, B-B shot.

Jaw swollen to size of a good case of mumps.

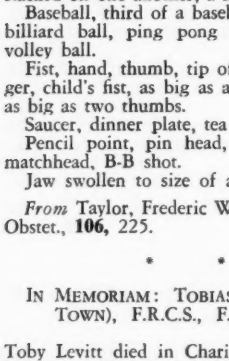
From Taylor, Frederic W. (1958): Surg. Gynecol. Obstet., **106,** 225.

* * *

IN MEMORIAM: TOBIAS LEVITT, M.A. (CAPE TOWN), F.R.C.S., F.R.C.S.E., F.R.C.S.I.

Toby Levitt died in Charing Cross Hospital on 22 February 1958 in his 49th year.

He was a distinguished student at the University of Cape Town, where he will be remembered as an outstanding anatomist. His contemporaries will recall his genial personality and his inexhaustible ingenuity in the invention of mnemonics. He attained first class honours in both anatomy and in physiology, before continuing with his medical education at the Middlesex Hospital, London, where he qualified in 1934. He served as a surgical specialist in the R.A.M.C. in World War II, and afterwards became Surgical First Assistant at the Thyroid Clinic at the New End Hospital, Hampstead.



Dr. T. Levitt

As has been the case with so many graduates from the Medical School in Cape Town, he made his mark in endocrinology, his researches in this field culminating in the publication (in 1954) of a considerable volume, entitled *The Thyroid: A Physiological, Pathological, Clinical and Surgical Study.* This book provoked world-wide interest and discussion and was characterized by the scrupulous attention to detail which was typical of his brilliant teaching.

In 1952 he was appointed a Hunterian Professor of the Royal College of Surgeons. His lecture was on *The Status of Lymphadenoid Goitre, Hashimoto's Disease and Riedel's Disease.* His address has been described as 'a model of its kind'. He held his audience spellbound on that occasion.

In 1952 he was the victim of an attack of coronary thrombosis, from which he suffered during an extensive lecture tour in America, and which brought about his final illness.

At the time of his death he was at work on a book on malignant disease of the thyroid gland.

Toby Levitt was well-known to many as a Lecturer in Surgery to the Fellowship of Post-Graduate Medicine. Students from all over the Commonwealth, and particularly India, appreciated his generosity as well as the endless trouble he took with his teaching.

His loss will be lamented by friends and colleagues all over the world.

* * *

MCNEIL LABORATORIES INCORPORATED

McNeil Laboratories announce that they are undertaking a large scale expansion programme. With this object in view they have purchased a large tract of land near Fort Washington, U.S.A., where they will erect a new headquarters building covering some 200,000 square feet. This will house the manufacturing laboratories, research departments and administrative offices and will require the employment of an additional 200 specialized workers to staff it. The new building is expected to be completed in 1959.

This development is all the more remarkable in the light of the fact that only 10 years ago McNeil doubled in size the then existing building. These enlarged and modernized facilities have proved inadequate for their rapidly expanding operations. McNeil's products are now prescribed by doctors in 22 countries throughout the world, in addition to America, and their overseas markets are continually being enlarged.

The firm is now managed by the third generation of McNeils associated with the pharmaceutical industry. McNeil Laboratories, as it now operates, was founded by Robert Lincoln McNeil who has retired from day-to-day management of the business, but continues to serve as a member of the Board of Directors.

The agents for McNeil Laboratories in South Africa are Westdene Products (Pty.) Limited:
Johannesburg—23 Essanby House, 175 Jeppe Street.
Cape Town—408 Grand Parade Centre, Castle Street.
Durban—66/67 National Mutual Building, Smith Street.
Pretoria—210 Medical Centre, Pretorious Street.

* * *

XVITH INTERNATIONAL CONGRESS OF THE HISTORY OF MEDICINE

FACULTY OF MEDICINE, MONTPELLIER (22–28 SEPTEMBER 1958)

The XVIth International Congress of the History of Medicine will be held at the Faculty of Medicine of Montpellier from Monday 22 to Sunday 28 September 1958, under the general presidency of Monsieur le Doyen Giraud, Dean of the Faculty.

Registrations can be made with Monsieur le Professeur Turchini, Président of the Organization Committee, Faculty of Medicine, Montpellier (Hérault), (France).

The following subjects have been selected for attention:

1. *Connections between the School of Montpellier and the Medical Institutions of Various Countries throughout the Centuries.*
2. *History and Expansion of Hospital Establishments.*
3. *Medical Iconography during the XVIIth Century.*

4. *The New World's Contribution to Therapeutics.*
5. *Varia.*

Entrance fees are:

6.000 francs (French) for non-members of the History of Medicine Society:
4.000 francs (French) for Membership; and to
3.500 francs (French) for persons accompanying participants.

The Organization Committee would be grateful to have the title of the communication a participant intends to present (10 minutes for each), as well as a short abstract.

Remittances may be made by cheque either to: *Compagnie Algérienne de Montpellier, No. W.*12–900–3; *or* to

Compte Courant Postal Montpellier No. 1178–03 (*XVI*e *Congrès International d'Histoire de la Médecine, Faculté de Médecine, Montpellier*).

Further Information from: The Secretary General: Dr. Louis Dulieu, M.D.

* * *

A NEW WEAPON IN THE WAR AGAINST LEPROSY

Leprosy—like malaria, plague, smallpox, and cholera—is a scourge that is common throughout wide areas of the globe. It is a chronic infectious disease which generally drags on for decades, causing conspicuous damage to the skin, nerves, and bones. The leprosy bacillus was first described in 1874, by the Norwegian Hansen. Fear of infection and disfigurement led to the banishment of lepers from society and to their confinement in leper colonies. At certain periods during the Middle Ages, there were tens of thousands of leper asylums in Europe; and, although the disease nowadays occurs chiefly in tropical countries, there is still a large number of institutes for lepers in other regions—including places as far north as Reykjavik in Iceland.

Quite apart from its medical significance, leprosy is first and foremost a social problem. Since the law usually insists on the segregation of lepers, the result is that sufferers seek to conceal their condition for as long as possible. This accounts for the fact that at present the number of reported cases of leprosy amounts to only about 600,000, whereas the actual total of victims is estimated at over 10 million. The struggle against this disease is thus one of the most urgent tasks confronting medical science to-day.

Hence great importance attaches to a report, published in the British journal *Leprosy Review*, on the great progress made in recent years in the treatment of leprosy. Nigeria in West Africa offers a good example of the fine work that has been achieved in controlling leprosy, thanks to close co-operation between Government officials, WHO and UNICEF, as well as missionaries. Besides the use of appropriate drugs, other steps taken have included the training of native inhabitants in the application of treatment, instruction in hygiene and the prevention of disease, and encouragement to victims of leprosy to come for treatment. Attention is now centred upon a new drug with which successful trials have been conducted by Dr. T. F. Davey, head of a research unit in Uzuakoli, Eastern Nigeria, supported by the British Leprosy Relief Association. The drug is a diphenyl thiourea compound, which was developed in the Ciba Research Laboratories in Switzerland and the United States.

Dr. Davey, whose findings have been corroborated by other research teams, reports that with this drug, known as CIBA 1906, he has obtained very good results in cases of leprosy. Experience has shown that it has various advantages over other forms of treatment used hitherto. It prevents scarring and disfigurement of the patient's skin and is also particularly valuable for treating children.

Dr. Davey describes it as 'an antileprosy drug of considerable potential importance with outstanding qualities.' In view of work now being done in the leprosy institutes, of the efforts being made by WHO, UNICEF and other organizations, and of experience already gained with this new drug, the Editor of *Leprosy Review* thinks that in certain areas there are good chances of eradicating this age-old scourge of humanity within a generation.

PREPARATE EN TOESTELLE

VESPARAX

Die vraag ‚Wat is slaap?' is nog nie beantwoord nie, want die neurofisiologiese meganisme wat die mens van die wakkere na die slapende toestand laat oorgaan, is nog steeds onopgelos, en die totstandbrenging van wat ons ‚slaap' noem, bly 'n geheim.

Barbituraat-slaap, hoewel dit doeltreffend is, het nog nie die probleem van nablywende effekte en die gebrek aan emosionele ontspanning tydens die slaap wat aldus veroorsaak word, opgelos nie.

Vesparax, 'n produk van Union Chimique Belge, bring 'n soort slaap teweeg wat baie nou aan natuurlike slaap verwant is.

Vesparax bevat:

Sekobarbitaal—'n kragtige, vinnigwerkende barbituraat.

Brallobarbitaal—'n progressief werkende barbituraat met 'n effek van middelmatige duur.

Aterax—'n doeltreffende kalmeerder wat die neiging om drome en nagmerries te hê, teëwerk.

Die samestelling van die twee barbiturate en *Aterax* in *Vesparax* onderbreek die psigogevoelsprikkels wat dikwels slaaploosheid veroorsaak, en bring 'n psigiese kalmte teweeg soos barbiturate alleen dit nie kan doen nie.

Dosis: Pasiënte was gewoond aan barbiturate is: 1 volle tablet voordat hulle gaan slaap; pasiënte wat gewoonlik nie slaapmiddels neem nie—½-tablet voor slapenstyd.

Verpakking: Buisies van 10 tablette en bottels van 100 tablette.

Verspreiders: Scherag (Pty.) Limited, Posbus 7539, Johannesburg.

SPARINE (PROMASIENHIDROCHLORIED)

'N NUWE NUTTIGE DOSISVORM

Sparine is van groot belang in psigiatriese noodgevalle, as die pasiënt mislik is of opbring, en vir verloskundige en chirurgiese kalmering.

Dit is tans verkrygbaar 'n stroop- sowel as tabletvorm.

Die nuttigheid daarvan word dus uitgebrei tot dit gebied van die kindergeneeskunde, en dit kan nou ook voorgeskryf word vir die volwasse pasiënt wat onwillig of nie in staat is om tablette in te sluk nie.

Beskikbaarstelling: Sparine-stroop—10 mg. per teelepelvol (5 k.s.). Bottels van 4 vloeistof-ons.

Sparine-tablette—25 mg., 100 mg. Bottels van 50.

Sparine-inspuiting—50 mg. per k.s. Flessies van 10 k.s.

Alleenverspreiders in Suid-Afrika: Wyeth Laboratories, Posbus 8138, Johannesburg, en Posbus 844, Oos-Londen.

CARBONET

'N NUWE NIE-VASKLEWENDE VERBINDSEL

Carbonet, 'n nuwe soort nie-vasklewende verbindsel geproduseer deur Smith & Nephew, Ltd., is 'n gewysigde vorm van *Jelonet*, waarin die konvensionele petroleumjellie-massa vervang is deur 'n in water oplosbare massa saamgestel uit 'n mengsel van poliëtileenglikols.

In vergelyking met die konvensionele paraffiengaasverbindsel B.P.C. (‚tulle gras') het *Carbonet* talle voordele. Een van laasgenoemde se belangrikste kenmerke is die feit dat wonde onder die verbindsel nie deurweek raak nie. Gevolglik word genesing nie vertraag nie.

Carbonet is aan uitvoerige kliniese toetse in 11 sentrums in die Verenigde Koninkryk onderwerp, en het die bewys gelewer dat dit 'n eersteklas verbindsel is. Al die geneeshere wat die verbindsel gebruik het, het *Carbonet* eenparig goedgekeur, en die mening uitgespreek dat dit in alle opsigte beter as paraffiengaasverbindsel is. Die volgende twee verslae is tipies van die resultate van die proefnemings.

Die eerste is afkomstig van die Ongevalleafdeling van die Stadshospitaal in St. Albans, Hertfordshire:

‚*Carbonet* is in meer as 20 gevalle gebruik vir die behandeling van skaafplekke, skoon sowel as besmet, vir brandwonde wat deur droë of nat hitte veroorsaak is, en vir ander oop wonde, soos dié wat deur spykers nagelaat word nadat die spyker self verwyder is. Die indrukke wat ek gekry het van die hoedanighede daarvan, in vergelyking met paraffiengaas, kan soos volg saamgevat word:

1. Daar is bevind dat *Carbonet* nie so maklik aan etterende wonde vaskleef nie.

2. *Corbonet* maak die omliggende vel nie so nat nie, en stel die wond in staat om gouer droog te word.

3. In een geval waar 'n allergiese uitslag deur paraffiengaas veroorsaak is, het die gebruik van *Carbonet* geen reaksie tot gevolg gehad nie, en die uitslag het gou-gou verdwyn.

Aan die hand van hierdie resultate het ek tot die gevolgtrekking geraak dat *Carbonet* 'n beter verbindsel as paraffiengaas is.

Tussen hakies, ek het bevind dat dit ook 'n besonder geskikte verbindsel is as dit oor 'n antibiotiese salf op 'n besmette wond aangelê word.' . . . L.R.C.P., M.R.C.S.

Die tweede verslag is afkomstig van St. Georgehospitaal, die bekende onderrighospitaal in Londen:

‚*Carbonet* is as aanvanklike basiese verbindsel vir die gewone chirurgiese wonde van 27 pasiënte gebruik. Dit was hoofsaaklik buikwonde, maar onder hulle was daar ook 'n aantal radikale mastektomieën skildkliergevalle.

Die tegniek was om die *Carbonet* aan te lê, om dit met 'n dik laag gewone gaas te bedek, en om die verbindsel dan met poreuse Elastoplast in posisie te bevestig. In geen enkele geval het sepse voorgekom nie, en by die eerste verbinding is daar bevind dat die wonde nie vetterig of deurweek was nie. By verwydering het die verbindsel nie aan die wonde vasgekleef nie, en dit het geskyn asof geen pyn veroorsaak is deur die vashaak van die maas aan die heg-tingsmateriaal nie.

Volgens my mening is *Carbonet* die beste nievasklewende verbindsel vir gewone chirurgiese wonde wat ek nog op die proef gestel het.' . . . M.S., F.R.C.S.

Carbonet is verkrygbaar in blikkies bevattende 36 steriele verbindsels, 3¾ duim x 3¾ duim groot, en in blikkies bevattende 'n lang steriele strook materiaal, 7½ duim breed en 4 jaart lank.

Nadere inligting is verkrygbaar van: Smith & Nephew (Pty.) Ltd., Posbus 92, Pinetown, Natal.

TETREX INTRAMUSCULAR (250 MG.)

Bristolabs (Pty.) Limited kondig die beskikbaarstelling aan van *Tetrex* Intramuscular (250 mg.).

Beskrywing: Vir die eerste keer voorsien *Tetrex* Intramuscular (250 mg.) die geneesheer nou van tetrasiklien in 'n praktiese binnespierse dosisvorm wat 'n antibiotiese bloedpeil na 'n enkele inspuiting produseer en hierdie peil 24 uur lank in stand hou. Dit word moontlik gemaak deur die unieke oplossingseienskappe van die tetrasiklienfosfaatkompleks. M.a.w. die geneesheer kan maksimale tetrasiklienbedrywigheid verseker deur 'n minimale hoeveelheid van die middel in te spuit. Uitgebreide kliniese ondervinding het aangetoon dat sodanige *Tetrex*-

inspuitings goed verdra word en dat daar betreklik min ongerief is.

Indikasies: Vir aanvanklike terapie en totdat mondelinge behandeling met *Tetrex*-kapsules of -stroop ingestel kan word. *Tetrex* Intramuscular met xilokaïen word aanbeveel vir baie van die gewone infeksies wat deur tetrasiklien-sensitiewe organismes veroorsaak word.

Onder die indikasies vir *Tetrex* Intramuscular is daar die volgende infeksies wat aan tetrasiklien-vatbare organismes te wyte is:

Infeksies van die Asemhalingskanaal: pneumonie, akute brongitis, faringitis, sinusontsteking, septiese seerkeel, kinkhoes.

Infeksies van die Urinêre Kanaal: Nier- en nierbekkenonsteking, nierbekkenontsteking, blaasontsteking, ontsteking van die prostataklier, urinebuisontsteking.

Infeksies van die Spysverteringskanaal: basillêre en amebiese disenterie, bakteriese diarree, maag- en derrmkatar.

Dermatologiese Infeksies: weefselontsteking, furinkulose, etterende huidontsteking, aknee.

Rickettsia- en Virusinfeksies: tifuskoors, trachoom, lymphogranuloma venereum en papegaaisiekte.

Profilakse tydens Chirurgie en Verloskunde: voor of volgende op chirurgie in nie-steriele streke.

Dosis: *Volwassenes*: die gewone minimum-dosis per dag is 250 mg., toegedien een maal in 24 uur. In die geval van ernstige infeksies kan dit nodig wees om 250 mg. al om die 12 uur in te spuit.

Kinders: Die dosis moet aangepas word by die liggaamsgewig en die toestand wat behandel word. Kliniese ondervinding het bewys dat die daaglikse dosis vir suigelinge en jong kinders 15–25 mg. per kg. liggaamsgewig behoort te wees, met 'n maksimum van 250 mg. per inspuiting op 'n enkele dag. Ouer kinders en kinders wat swaarder is, het ten minste 250 mg. per dag nodig, na gelang van die kliniese oordeel van die geneesheer.

Tetrex Intramuscular behoort diep in die boudspier ingespuit te word.

Aanbieding: Iedere flessie bevat 250 mg. tetrasiklienfosfaat-kompleks, en behoort voor inspuiting met 1.6 k.s. steriele water of normale soutoplossing gerekonstitueer te word.

Fabrikante: Bristol Laboratories Inc., Syracuse, New York.

S.A. Verspreiders: Bristolabs (Pty.) Limited, Posbus 2515, Johannesburg.

FERSAMAL (GLAXO)

FERROFUMARAAT-TABLETTE

Beskrywing: Klein, bruin, smaaklike tablette sonder buitelaag.

Samestelling: Elke tablet bevat 200 mg. watervrye ferrofumaraat. Dit verskaf ongeveer 65 mg. ferroyster—gelyksoortig aan die konvensionele ferrosulfaat-tablette van 3 grein.

Uitwerking: Yster wat in die vorm van ferrofumaraat ingeneem word, word goed ge-absorbeer en benut. By die behandeling van bloedarmoede weens ystertekorte, val die hemoglobien-reaksie binne die aanvaarde kader.

Aanwysings: Vir voorkoming en behandeling van bloedarmoede weens ystertekorte.

Voordele: Fersamal word goed verdra en maag- of dermnewe-effekte kom uiters selde voor. Die klein tablette sonder buitelaag kan maklik ingesluk word. Gevolglik word die medewerking van die pasiënt gewoonlik verkry en verseker dit doeltreffende behandeling.

Dit is onwaarskynlik dat klein kinders die tablette aanloklik sal vind, maar selfs al sou hulle per ongeluk daarvan insluk, is dit onwaarskynlik dat dit enige ernstige nadelige uitwerking sal hê.

Dosismaat: Een tablet driekeer per dag verskaf 'n bevredigende hemoglobienreaksie; indien verlang kan twee tablette egter toegedien word. Die tablette kan maklik ingesluk word, maar indien verkies, kan hulle ook fyngemaak of gekou word.

Verpakking: Buisie van 100.

Glaxo Laboratories (S.A.) (Pty) Ltd, Manchesterweg, Wadeville, Transvaal.

MIDICEL-TABLETTE

Parke, Davis, kondig die beskikbaarstelling aan van *Midicel*, 'n nuwe sulfonamied met 'n langdurige effek, wat talle gram-negatiewe en gram-positiewe bakteriese infeksies met welslae bestry. Kliniese toetse het bewys dat *Midicel* veral geskik is vir die behandeling van pasiënte wat aan infeksies van die geslags-urinêre kanaal ly.

Beskrywing: *Midicel* (sulfametoksipiridasien, Parke, Davis) is chemies bekend as 3-sulfanilamied-6-metoksipiridasien, en is 'n sulfonamied wat vinnig geabsorbeer en stadig afgeskei word. Om doelmatige bloedkonsentrasies in stand te hou, moet pasiënte wat met die konvensionele sulfonamiedpreparate behandel word, hulle by die allerstrengste dosisvoorskrifte bepaal—gewoonlik 'n dosis al om die 4 of 6 uur. Sulke lastige dosisreëls bring dikwels mee dat dosisse oorgeslaan word, met die gevolg dat daar tydperke van lae bloedkonsentrasies is, in die loop waarvan die bakterieë baie vinnig vermenigvuldig. *Midicel* is die vervulling van 'n strewe wat lank reeds met sulfonamiedterapie beoog word, nl. 'n veilige mikrobebestrydende middel wat vinnig geabsorbeer word, 'n langdurige uitwerking het, en 'n volgehoue terapeutiese effek uitoefen as klein mondelinge dosisse geneem word. 'n Enkele daaglikse dosis verskaf doeltreffende bloedpeile gedurende 'n tydperk van meer as 24 uur, en skakel die gevaar van ‚bloeddaling' ten gevolge van oorgeslaande dosisse heeltemal uit.

Indikasies: Die getuienis wat tot dusver ingewin is, dui daarop dat *Midicel* klinies doeltreffend is vir die behandeling van 'n groot verskeidenheid van infeksies wat op sulfonamiedterapie reageer. Hulle sluit in asemhalingsinfeksies,[1] infeksies van die geslags-urinêre kanaal,[2-5] longabsesse,[5] bakteriese disenterie,[1] rumatiekkoors[6] en asimptomatiese basillurie by swanger vrouens.[4] In die geval van laasgenoemde twee kwale is *Midicel* as profilaktiese middel gebruik.

Die feit dat *Midicel* slegs middelmatig geasetiliseer is en stadig deur die niere afgeskei word, tesame met die baie maklike oplosbaarheid daarvan teen die suur-pH wat gewoonlik in gevalle van nierinfeksie aangetref word, verduidelik die doeltreffendheid van die middel vir die behandeling van infeksies van die geslags-urinêre kanaal wat veroorsaak word deur *Esch. coli, Aerobacter aerogenes,* streptokokki, stafilokokki, Gram-negatiewe basille, en difteroïede, asook sommige gevalle van Proteus-infeksie.

Dosis en Toediening: Aangesien *Midicel* langdurige, doeltreffende bloedpeile tot gevolg het, hoef slegs 'n baie klein gedeelte van die gewone sulfonamieddosis voorgeskryf te word. Die aanbevole dosis vir volwassenes in gevalle van ligte tot middelmatig ernstige infeksies is 2 tablette (1 g.) op die eerste dag, dan 1 tablet (0.5 g.) daagliks, of 2 tablette al om die ander dag. In die geval van ernstige infeksies word 4 tablette (2 g.) op die eerste dag, en dan die gewone 1 tablet (0.5 g.) per dag aanbeveel.

Die dosis vir kinders moet by hul liggaamsgewig aangepas word. Vir iedere 20 pond, ¼-tablet, 0.125 g. (of 6·25 mg. per pond liggaamsgewig) op die eerste dag; dan ¼-tablet, 0.0625 g. (of 3.12 mg. per pond liggaamsgewig) per dag.

Met die oog op die stadige afskeiding en die langdurige bloedpeile word daar aanbeveel dat hierdie dosisse nie oorskry moet word nie. Hou 5–7 dae lank daarmee vol, of totdat die pasiënt oor 'n tydperk van 48–72 uur geen simptome openbaar nie.

Voorsorgsmaatreëls: Die newe-effekte wat oor die algemeen voorkom by pasiënte wat met sulfonamiede behandel word, kan soms ook hul verskyning maak as die geneesheer *Midicel* voorskryf. Onder hierdie newe-effekte is daar hoofpyn, malaise, anoreksie, 'n huiduitslag en medisynekoors. Weens die klein dosisse en die geringe konsentrasies in die gesonde nier is kristallurie onwaarskynlik, maar 'n doeltreffende hoeveelheid vloeistof moet ingeenem word tydens en gedurende ten minste 24–48 uur ná behandeling. Pasiënte met defekte nierfunksie moet sorgvuldig dopgehou word, want buitensporige akkumulasies van die middel is moontlik.

Inligting oor Verpakking: Tablette van 0.5 g. wat in vier kwarte gekeep is. Flessies van 12, en bottels van 100.

VERWYSINGS

1. Ross, S., Ahrens, W. E. en Zaremba, E. A. (1957): Ann. N.Y. Acad. Sci., **69**, 501.
2. Frisk, A. R. en Wassen, A. (1957): *Antibiotics* Annual, 1956–1957, bl. 424, Medical Encyclopedia, Inc., New York.
3. Jackson, G. G. en Grieble, H. G. (1957): Ann. N.Y. Acad. Sci., **69**, 493.
4. Jones, W. en Finland, M. (1957): Ann. N.Y. Acad. Sci., **69**, 509.
5. Walker, W. F. en Hamburger, M. (1957): Ann. N.Y. Acad. Sci., **69**, 509.
6. Lepper, M. H., Simon, A. J. en Marienfeld, C. J. (1957): Ann. N.Y. Acad. Sci., **69**, 485.

OTAMIDYL-OORDRUPPELS

Maybaker (S.A.) (Pty.) Ltd. kondig die beskikbaarstelling aan van *Otamidyl*, 'n bakterie- en swambestrydende oordruppel.

Otamidyl bevat dibromopropamidienisetionaat 0.15% g/v en diamidinodifenielamiendihidrochloried 0.5% g/v in propileenglikol.

Hierdie samestelling behou sy bakterie- en swambestrydende eienskappe in die aanwesigheid van etter, bloed en weefselafval. Organisme-soorte wat weerstand teen antibiotica bied, is volkome gevoelig vir die diamidiene.

Otamidyl word aangedui vir plaaslike behandeling in gevalle van otitis externa en chroniese etterende otitis media.

BECANTYL

VIR DIE BESTRYDING VAN 'N NIKS-OPLEWERENDE GEHOES

Becantyl, die aktiewe bestanddeel waarvan natrium 2:6 ditersiêrbutielnaftaleenmonosulfonaat is, is 'n hoesmiddel wat klinies die bewys gelewer het dat dit doeltreffend vir die bestryding van 'n niksoplewerende gehoes is.

Die aktiewe bestanddeel is nie verwant aan morfien of guajakol nie, en het geeneen van die newe-effekte wat met hierdie stowwe geassosieer is nie.

Die afwesigheid van lomerigheid, anoreksie of hardlywigheid maak *Becantyl* die verkieslike middel vir die behandeling van 'n gehoes by kinders of ou mense. Die terapeutiese veiligheidsgrens is groot.

Becantyl is verkrygbaar in twee vorms—as 'n stroop, en as tablette. Die stroop word verpak in bottels wat 4 vloeistof-ons bevat, die tablette in dosies van 24, toegedraai in goudblad.

Dosis: Die aanbevole dosis *Becantyl* is:

Stroop:

Volwassenes: 2 teelepelsvol.

Kinders: 3–6, ½-teelepelvol; 7–15 jaar, ½–1 teepelvol, 3 maal per dag, of soos voorgeskryf.

Tablette:

Volwassenes: 2 tablete 3 of 4 maal per dag, of soos voorgeskryf.

Kinders: Die dosis behoort dienooreenkomstig gewysig te word.

Die tablette moet heel ingesluk word.

Fabrikante: Horlicks Limited, Slough, Buckinghamshire.

Alleenverspreiders vir Suid-Afrika: B.P.D. (S.A.) (Pty.) Ltd., Posbus 45, Jeppestown, Transvaal.

TRILAFON-INSPUITING

SCHERING CORPORATION, V.S.A

Beskrywing: Iedere k.s. *Trilafon*-inspuiting bevat 5 mg. perfenasien, 'n besonder kragtige kalmerings- en brakingbestrydingsmiddel in 'n waterige draer vir binnespierse of binne-aarse inspuiting.

Voordele: 'n Gunstige terapeutiese verhouding word deur *Trilafon* bewerkstellig. Dit het 'n parenterale milligram-krag wat veel groter as dié

van ander fenotiasien-samestellings is, sonder 'n ooreenstemmende vermeerdering van die outonomiese, hematologiese of lewer-newe-effekte. Kleiner dosisse kan derhalwe voorgeskryf word nie alleen vir pasiënte wat reeds met die ouer middels behandel is nie, maar ook vir pasiënte wat nog nie tevore behandel is nie.

Die mate van adrenergiese versperring wat deur *Trilafon*-inspuiting veroorsaak word, is veel kleiner as dié wat 'n mens met sekere ander fenotiasien-kalmeermiddels teëkom. Betekenisvolle hipotensie is dus 'n uiters seldsame verskynsel. Diep binnespierse inspuiting word besonder goed verdra; geen plaaslike reaksies, insluitende pyn by die inspuitingsplek, is gerapporteer nie. Nóg agranulositose nóg geelsug het op die inspuiting van *Trilafon* gevolg.

Indikasies: *Trilafon*-inspuiting word aangedui as aanvanklike terapie of vir die behandeling van akute episodes by geestelik en emosioneel versteurde pasiënte; akute alkoholisme; mislikheid en braking wat aan verskillende oorsake te wyte is; verloskunde; 'n onbedaarlike gehik; kroniese pyn en ernstige puritus. Die kalmerings- en brakingbestrydingseffek van *Trilafon*-inspuiting is ook van groot waarde vir pasiënte wat operasies ondergaan.

Dosis en Toediening: *Trilafon*—Binnespiers. Die dosisse moet geïndividualiseer en aangepas word by die toestand wat behandel word, die erns van die simptome, en die pasiënt se reaksie. Die dosisse vir kinders is nog nie vasgestel nie. Kinders bo 12 jaar kan die klienste dosis wat vir volwassenes voorgeskryf word, ontvang.

Die gewone aanvanklike dosis is 5 mg. (1 k.s.). Dit kan al om die 6 uur herhaal word. In gewone omstandighede moet die totale daaglikse dosis egter nie 15 mg. oorskry in die geval van pasiënte wat in staat is om rond te loop, of 30 mg. in die geval van bedlêende pasiënte nie. Indien dit nodig is om bevredigende beheer oor die simptome in ernstige toestande uit te oefen, kan 'n aanvanklike dosis van 10 mg. binnespiers toegedien word.

Die inspuiting moet geskied onderwyl die pasiënt sit of lê, en na die toediening van *Trilafon* moet die pasiënt 'n ruk lank dopgehou word. Duiseligheid of betekenisvolle hipotensie na 'n Trilafon-inspuiting is seldsame verskynsels.

Diep binnespierse inspuiting word besonder goed verdra; geen plaaslike reaksies is gerapporteer nie. 'n Terapeutiese effek kan gewoonlik binne tien minute waargeneem word, en bereik sy hoogtepunt na 1 of 2 uur. Die doeltreffende effek van die middel duur gewoonlik 6 uur lank, maar soms word hierdie tydperk tot 12 en selfs 24 uur verleng.

Pasiënte moet so gou moontlik na mondelinge terapie oorgaan. In die reel kan dit binne 24 uur geskied. In sommige gevalle was dit egter nodig om inspuitingsterapie maande lank toe te pas.

Daar is vasgestel dat *Trilafon*-inspuiting kragtiger as die tablette is. Derhalwe moet gelyke of groter dosisse voorgeskryf word wanneer die pasiënt na mondelinge terapie oorgaan.

Trilafon-inspuiting—Binne-aars. Die binne-aarse toediening van *Trilafon* word nie aanbeveel nie, behalwe in gevalle waar dit absoluut noodsaaklik is om ernstige braking, 'n onbedaarlike gehik of akute simptome soos braking tydens 'n operasie te beheer. As die binne-aarse roete gebruik word, moet groot sorg en versigtigheid aan die dag gelê word. Dit moet slegs toegepas word in die geval van bedlêende pasiënte in hospitale, en die dosis moet nie 5 mg. oorskry nie. As dit op hierdie manier gebruik word, moet die binne-aarse inspuiting in die vorm van 'n verdunde oplossing toegedien word ooreenkomstig die fraksionele inspuitings- of die stadige indruppingsmetode.

Verpakking: *Trilafon*-inspuiting, 5 mg., dosies van 6 ampulle.

Scherag (Pty.) Limited, Posbus, 7539, Johannesburg.

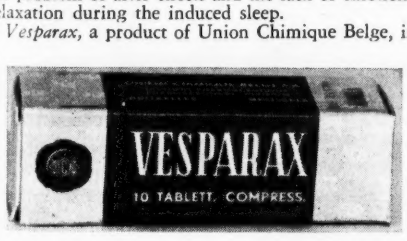
PREPARATIONS AND APPLIANCES

VESPARAX

The question ' what is sleep? ' has not been answered yet, for the neurophysiologic mechanism which causes Man to pass from wakefulness to sleep is still unsolved and the induction of what we call ' sleep ' remains a mystery.

Barbituric sleep, although effective, has not solved the problem of after effects and the lack of emotional relaxation during the induced sleep.

Vesparax, a product of Union Chimique Belge, induces sleep which very much resembles natural sleep.



Vesparax contains:

Secobarbital—A powerful, quick acting barbiturate.

Brallobarbital—A progressively acting barbiturate with medium length duration.

Aterax—An effective tranquillizer which inhibits the tendency to dreams and nightmares.

The combination of the two barbiturates and *Aterax* in *Vesparax* interrupts the psychoaffective stimuli which often cause insomnia, and brings about a psychic calm which barbiturates alone are unable to achieve.

Dosage: Patients accustomed to taking barbiturates —1 full tablet before retiring;

Patients who usually do without soporifics—½ tablet before retiring.

Packing: Tube of 10 tables and bottle of 100 tablets.

Distributors: Scherag (Pty.) Limited, P.O. Box 7539, Johannesburg.

SPARINE (PROMAZINE HYDROCHLORIDE)

A NEW USEFUL DOSAGE FORM

Sparine has important indications in psychiatric emergencies, in nausea and vomiting and for obstetrical and surgical sedation.

It is now available as a syrup as well as in tablet form.

Its usefulness is thus extended to the field of paediatrics as well as for the adult patient unable or unwilling to swallow tablets.

Supplied: *Sparine* syrup—10 mg. per teaspoonful (5 c.c.). Bottles of 4 fl. oz.

Sparine tablets—25 mg., 100 mg. Bottles of 50.

Sparine injection—50 mg. per c.c. Vials of 10 c.c.

Sole South African Distributors: Wyeth Laboratories, P.O. Box 8138, Johannesburg and P.O. Box 844, East London.

CARBONET—A NEW NON-ADHERENT DRESSING

Carbonet, a new type of non-adherent dressing produced by Smith & Nephew Ltd. is a modified form of *Jelonet*, in which the conventional petroleum jelly mass has been replaced by a water-soluble mass composed of a mixture of polyethylene glycols.

Carbonet has many advantages over the conventional paraffin gauze dressing B.P.C. (*tulle gras*), and one of its most important features is the fact that wounds do not become macerated beneath it. Therefore healing is not retarded.

Carbonet has been given an extensive clinical trial in 11 centres in the United Kingdom, and has proved itself to be a first class dressing. All the surgeons concerned were unanimous in their approval of *Carbonet*, and considered that it was better than paraffin gauze dressing B.P.C. in all respects. The following two reports are typical of the trials.

The first one comes from the Casualty Department of the St. Albans City Hospital in Hertfordshire:

'*Carbonet* was used in more than 20 cases for the treatment of abrasions, both clean and infected, scalds, burns and for other open wounds such as the nail bed following nail removal. The impressions given of its properties compared with paraffin gauze were as follows:

1. *Carbonet* was found to be less adherent to discharging wounds.

2. *Carbonet* made the surrounding skin less soggy and allowed the wound to dry more quickly.

3. In one case in which an allergic rash had been caused by paraffin gauze, *Carbonet* caused no reaction and the rash quickly disappeared.

From these results, I have come to the conclusion that *Carbonet* is a superior dressing to paraffin gauze.

Incidentally, I found it a very suitable dressing when applied over an antibiotic ointment on an infected wound.' . . . L.R.C.P., M.R.C.S.

The second report comes from the well-known London teaching hospital (St. George's):

'The *Carbonet* was applied as an initial basic dressing to ordinary surgical wounds in 27 patients. These were predominantly abdominal wounds but included radical mastectomy and thyroid cases as well.

The technique was to apply the *Carbonet*, cover this with a thick layer of ordinary gauze and then keep the dressing in place with porous Elastoplast. In no case did sepsis occur and the wounds on first dressings were never greasy or soggy. On removal, the dressings did not adhere to the wound, and there appeared to be no pain caused by catching of the mesh in the sutures.

In my opinion, *Carbonet* is the best non-adherent dressing I have tested for routine surgical wounds.' . . . M.S., F.R.C.S.

Carbonet is available in tins containing 36 sterile dressing measuring 3¾ inch x 3¾ inch, and in tins containing a continuous, sterile strip of material 7½ inch wide and 4 yards long.

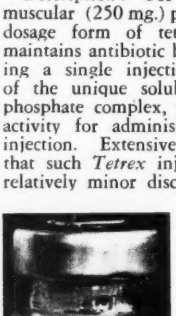
Further information can be obtained from:

Smith & Nephew (Pty.) Ltd., P.O. Box 92, Pinetown, Natal.

TETREX INTRAMUSCULAR (250 MG.)

Bristolabs (Pty.) Limited announce the introduction of *Tetrex* Intramuscular 250 mg.

Description: For the first time, *Tetrex* Intramuscular (250 mg.) provides a practical intramuscular dosage form of tetracycline which produces and maintains antibiotic blood levels for 24 hours following a single injection. This is possible, because of the unique solubility properties of tetracycline phosphate complex, permitting maximal tetracycline activity for administration in minimal volume of injection. Extensive clinical experience has shown that such *Tetrex* injections are well tolerated with relatively minor discomfort.



Indications: For initial therapy and until oral medication (*Tetrex* capsules or syrup) can be given. *Tetrex* Intramuscular with Xylocaine is recommended in many common infections due to tetracycline-sensitive organisms:

Indications for *Tetrex* Intramuscular include the following infections due to tetracycline-susceptible organisms:

Respiratory Tract Infection: pneumonia, acute bronchitis, pharyngitis, sinusitis, septic sore throat, whooping cough.

Urinary Tract Infections:

pyelonephritis, pyelitis, cystitis, prostatitis, urethritis.

Gastrointestinal Infections: bacillary and amoebic dysentery, bacterial diarrhoea, gastroenteritis.

Dermatological Infections: cellulitis, furunculosis, pustular dermatosis, acne.

Rickettsial and Viral Infections: typhus fever, trachoma, lymphogranuloma venereum and psittacosis.

Prophylaxis in Surgery and Obstetrics: prior to or following surgery in unsterile fields.

Dosage: *For the Adult*: the usual minimum daily dose is 250 mg. administered once every 24 hours. Severe infections may require injections of 250 mg. at 12-hour intervals.

For Children: Dosage should be adjusted according to body weight and conditions being treated. Clinical experience indicated that the daily dosage for infants and young children should be 15–25 mg. per Kg. of body weight up to a maximum of 250 mg. per single daily injection. Older and higher weight children require at least 250 mg. per day depending upon the clinical judgment of the physician.

Tetrex Intramuscular should be injected deeply into the gluteal muscle.

Presentation: Each vial contains Tetracycline Phosphate Complex 250 mg. and should be reconstituted with 1.6 c.c. of sterile water or normal saline before injection.

Manufacturers: Bristol Laboratories Inc., Syracuse, New York.

S.A. Distributors: Bristolabs (Pty.) Limited, P.O. Box 2515, Johannesburg.

FERSAMAL (GLAXO)

FERROUS FUMARATE TABLETS

Description: Small, brown, uncoated and palatable tablets.

Composition: Each tablet contains 200 mg. anhydrous ferrous fumarate. This provides approximately 65 mg. ferrous iron—similar to the conventional 3-grain ferrous sulphate tablet.

Mode of Action: Iron taken in the form of ferrous fumarate is well absorbed and utilized. In the treatment of iron deficiency anaemias, haemoglobin responses are well within the accepted range.

Indications: For prophylaxis and treatment of iron deficiency anaemias.

Advantages: Fersamal tablets are well tolerated and gastric or intestinal disturbance is extremely rare.

The small uncoated tablets are easily swallowed. This often secures co-operation of the patient and ensures effective therapy being taken.

The tablets are not likely to be attractive to small children but even if accidentally swallowed are unlikely to cause serious harmful effects.

Dosage: One tablet three times a day usually produces a satisfactory haemoglobin response; two tablets may be given if desired. The tablets are easily swallowed but may be crushed or chewed if preferred.

Packs: Tube of 100.

Glaxo Laboratories (S.A.) (Pty) Ltd, Manchester Road, Wadeville, Transvaal.

MIDICEL TABLETS

Parke, Davis, have introduced *Midicel*, a new long-acting sulphonamide, which effectively combats many gram-negative and gram-positive bacterial infections. Clinical experience has demonstrated *Midicel* to be particularly appropriate in treating patients with infections of the genito-urinary tract.

Description: *Midicel* (sulphamethoxypyridazine, Parke, Davis) is chemically known as 3-sulphanilamide-6-methoxypyridazine, and is a sulphonamide which is rapidly absorbed and slowly excreted. In order to maintain adequate blood concentrations, conventional sulphonamide preparations require faithful adherence to a rigid dosage schedule—usually a dose every 4 or 6 hours. Such a burdensome schedule frequently results in omitted doses and consequent periods of low blood concentrations during which bacteria may again multiply. *Midicel* represents the fulfilment of a long quest in sulphonamide therapy —a safe, rapidly absorbed, long-acting antimicrobial agent that provides a sustained therapeutic effect with low oral dosage. A single daily dose provides effective blood levels for more than 24 hours, eliminating the hazard of blood 'fall-off' due to the omitted doses.

Indications: Evidence to date indicates that *Midicel* is clinically effective in a variety of infections responsive to sulphonamide therapy. These include respiratory infections,[1] genito-urinary tract infections,[2-5] lung abscess,[5] bacterial dysentery,[1] rheumatic fever,[6] and asymptomatic bacilluria in pregnant women.[4] In the last two indications, *Midicel* has been employed prophylactically.

The fact that *Midicel* is only moderately acetylated and slowly excreted by the kidneys, together with its high solubility at the acid pH commonly observed in kidney infections, adequately explain its effectiveness in genito-urinary tract infections, due to *Esch. coli*, *Aerobacter aerogenes*, streptococci, staphylococci, gram-negative bacilli, diphtheroids, and some cases of Proteus infection.

Dosage and Administration: As *Midicel* produces prolonged effective levels in the blood it requires only a fraction of the dosage usual for most sulphonamides. The recommended adult dose in mild to moderately severe infections is 2 tablets (1 g.) the first day, then 1 tablet (0.5 g.) daily, or 2 tablets every other day. For severe infections, 4 tablets (2 g.) the first day, then the usual 1 tablet (0.5 g.) daily is recommended.

Children require dosage in proportion to body weight. For each 20 lb. ¼ tablet, 0.125 g. (or 6.25 mg. per lb. body weight) the first day; then ⅛ tablet, 0.0625 g. (or 3.12 mg. per lb. body weight) daily.

Because of slow excretion rates and prolonged blood levels, it is recommended that this dosage should not be exceeded. Continue for 5–7 days or until patient is free of symptoms for 48–72 hours.

Precautions: Side effects common to the sulphonamides in general may sometimes occur with *Midicel*. These include headache, malaise, anorexia, skin rash and drug fever. Owing to low dosage and low concentration in the healthy kidney, crystalluria is unlikely, but adequate fluid intake should be maintained during and for at least 24–48 hours after treatment. Patients with impaired renal function should be followed closely since excessive accumulations of the drug may occur.

Package Information: Quarter scored tablets of 0.5 g. in vials of 12 and bottles of 100.

REFERENCES

1. Ross, S., Ahrens, W. E. and Zaremba, E. A. (1957): Ann. N.Y. Acad. Sci., **69,** 501.
2. Frisk, A. R. and Wassen, A. (1957): *Antibiotics Annual,* 1956–1957, p. 424 Medical Encyclopaedia, Inc., New York.
3. Jackson, G. G. and Grieble, H. G. (1957): Ann. N.Y. Acad. Sci., **69,** 493.
4. Jones, W. and Finland, M. (1957): Ann. N.Y. Acad. Sci., **69,** 473.
5. Walker, W. F. and Hamburger, M. (1957): Ann. N.Y. Acad. Sci., **69,** 509.
6. Lepper, M. H., Simon, A. J. and Marienfeld, C. J. (1957): Ann N.Y. Acad. Sci., **69,** 485.

OTAMIDYL EAR DROPS

Maybaker (S.A.) (Pty.) Ltd., announce the introduction of *Otamidyl* brand antibacterial and antifungal ear drops.

Otamidyl contains dibromopropamidine isethionate 0.15% w/v and diamidinodiphenylamine dihydrochloride 0.5% w/v in propylene glycol.

The antibacterial and antifungal properties of these compounds are retained in the presence of pus, blood, and tissue debris. Strains of organisms which are resistant to antibiotics are fully sensitive to the diamidines.

Otamidyl is indicated for use by topical application in the treatment of otitis externa and chronic suppurative otitis media.

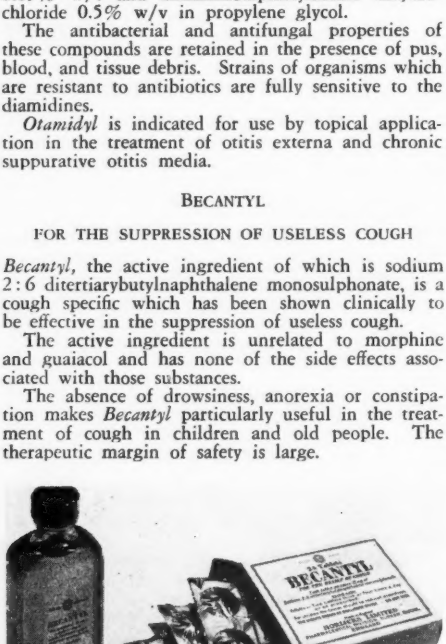
BECANTYL

FOR THE SUPPRESSION OF USELESS COUGH

Becantyl, the active ingredient of which is sodium 2:6 ditertiarybutylnaphthalene monosulphonate, is a cough specific which has been shown clinically to be effective in the suppression of useless cough.

The active ingredient is unrelated to morphine and guaiacol and has none of the side effects associated with those substances.

The absence of drowsiness, anorexia or constipation makes *Becantyl* particularly useful in the treatment of cough in children and old people. The therapeutic margin of safety is large.



Becantyl is available in two forms, as a syrup and as tablets. The syrup is packed in 4 fl. oz. bottles; the tablets in boxes of 24 in gold foil strips.

Dose: The recommended doses for *Becantyl* are:

Syrup:

Adults: 2 teaspoonfuls.

Children: 3–6 years: ½ teaspoonful; 7–15 years: ½–1 teaspoonful, 3 times a day or as prescribed.

Tablets:

Adults: 2 tablets, 3 or 4 times a day, or as prescribed.

Children: The dosage should be varied accordingly.

The tablets should be swallowed whole.

Manufacturers: Horlicks Limited, Slough, Buckinghamshire.

Sole South African Distributors: B.P.D. (S.A.) (Pty.) Ltd., P.O. Box 45, Jeppestown, Transvaal.

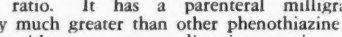
TRILAFON INJECTION

SCHERING CORPORATION, U.S.A.

Description: Each c.c. of *Trilafon* Injection contains 5 mg. perphenazine, an extremely potent tranquillizing and anti-emetic drug, in an aqueous vehicle for intramuscular or intravenous injection.

Advantages: *Trilafon* provides a favourable therapeutic ratio. It has a parenteral milligramme potency much greater than other phenothiazine compounds without a corresponding increase in autonomic, haematologic or hepatic side effects. Smaller doses are therefore required both in patients under treatment with older drugs and in those not previously treated.

The degree of adrenergic blockade with *Trilafon* Injection is considerably less than may be encountered with certain other phenothiazine tranquillizers. Thus significant hypotension has rarely been reported. Deep intramuscular injection is extremely well tolerated; no local reactions, including pain at the site of injection, have been reported. Neither agranulocytosis nor jaundice has been reported with *Trilafon* Injection.



Indications: *Trilafon* Injection is indicated as initial therapy or in the treatment of acute episodes in agitated mental and emotional disturbances; acute alcoholism; nausea and vomiting due to various causes; obstetrics; hiccoughs; chronic pain and severe pruritus. The tranquillizing and anti-emetic effects of *Trilafon* Injection are also valuable in surgery.

Dosage and Administration: *Trilafon*—Intramuscular. Dosage should be individualized and adjusted according to the condition treated, severity of symptoms and patient's response. Paediatric dosage has not yet been established. Children over 12 may receive the lowest limit of adult dosage.

The usual initial dose is 5 mg. (1 c.c.). This may be repeated every 6 hours. The total daily dosage ordinarily should not exceed 15 mg. in ambulatory or 30 mg. in hospitalized patients. When required to achieve satisfactory control of symptoms in severe conditions, an initial 10 mg. intramuscular dose may be given.

The injection should be given with the patient seated or recumbent and the patient should be observed for a short period after administration. Dizziness or significant hypotension after *Trilafon* Injection is a rare occurrence.

Deep intramuscular injection is extremely well tolerated; no local reactions have been reported. Therapeutic effect is usually evidenced in ten minutes and is maximal in 1 to 2 hours. The average duration of effective action is 6 hours and occasionally 12 to 24 hours.

Patients should be placed on oral therapy as soon as practicable. Generally, this may be achieved within 24 hours. However, in some instances, patients have been maintained on injectable therapy for several months.

It has been established that *Trilafon* Injection is more potent than tablets; therefore, equal or higher dosage should be used when the patient is transferred to oral therapy.

Trilafon Injection—Intravenous. Intravenous administration of *Trilafon* Injection is not recommended except when absolutely necessary to control severe vomiting, intractable hiccoughs, or acute symptoms such as violent retching during surgery. This route of administration should be used with particular caution and care, and should be limited to recumbent, hospitalized adults and to doses not exceeding 5 mg. When employed in this manner, intravenous injection ordinarily should be given as a diluted solution using either fractional injection or a slow drip infusion.

Packing: *Trilafon* Injection, 5 mg. Boxes of 6 Ampoules.

Scherag (Pty.) Limited, P.O. Box 7539, Johannesburg.

CORRESPONDENCE

A PROPOSED INSTITUTE FOR THE STUDY OF MAN IN AFRICA

TO COMMEMORATE THE LIFE AND WORK OF PROF. RAYMOND ARTHUR DART

To the Editor: At the end of 1958, Prof. R. A. Dart retires from the Chair of Anatomy at the University of the Witwatersrand, Johannesburg—a position which he has filled with lustre for 35 years. It would fill many pages of this *Journal* to sketch, even in outline, all that the medical, dental and allied professions owe to him in South Africa; nor does space permit even a cursory survey of his immense contributions to research on the origin of Man and on modern Man in Africa. However, it would not be out of place to refer here to the stimulation Professor Dart has given to the great numbers of his colleagues and the students who have passed through his hands. It is in this role—as a maker of men, as one who always elicited the best from his students and associates—that Professor Dart gave his finest gift to a young South Africa: in which the medical, dental and nursing professions were budding and those of physiotherapy and occupational therapy not yet conceived; in which research was emerging from its cocoon and in which the immense fruits of the sub-continent were awaiting the fertilizing impact, not only of the savants from abroad, but also of our own graduates, equipped and inspired to promote the advancement of knowledge.

Among the members and research students of his Department, who have subsequently achieved distinction, may be mentioned the late Dr. Gordon Laing, the late Prof. L. R. Shore, Profs. Alexander Galloway and Lawrence H. Wells, the brothers Gear, Profs. Joseph and Theodore Gillman, Dr. J. S. Weiner, Dr G. W. H. Schepers and Prof. J. C. Middleton Shaw. It is well known that the first Dental Faculty in South Africa stems directly from Professor Dart's appointment of Dr. Shaw as a Senior Lecturer in Comparative Odontology in the Department of Anatomy.

It is with such thoughts in mind that a group of medical and dental men, past and present students of Professor Dart, have conceived the idea of commemorating his work by the proposed establishment of an Institute for the Study of Man in Africa. Briefly, the objectives would be as follows:

Primarily, the Institute would aim to advance the study of the living peoples of Africa in health and disease. This would embrace bodily structure, function and pathology; diet and nutrition; tropical diseases and other medical problems; physical anthropology, heredity and racial composition; the climatic adaptations of Man at the equator, in the tropics, and in the sub-tropics, in the forests, deserts and mountain regions; dental eruption, attrition and decay. So much for the physical well-being of the people. This leads on to the psycho-social make-up of the peoples of Africa and their attitudes towards illness, as part of their general pattern of cultures, including also art, music and languages, tribal and kinship structure, and population problems.

Secondly, the Institute would try to foster the study of Man's ancestry in Africa—the fossil men, their evolution and migrations and hybridizations, the animals and plants and the changing climates associated with them. Included here, too, would be a study of their way of life, stone and bone implements, burial customs and artistic creations.

South Africa would seem to be ideally situated for the establishment of a continentally important Institute of this character and, within the Union, no better position than Johannesburg could be found. Here, within a narrow radius, are to be found the fossil ape-men, the *Australopithecinae* from Sterkfontein, Taung and Makapansgat; the Bushmen, those living fossils whose straggling remnants occur at Lake Chrissie to the east and in the Kalahari to the west, of Johannesburg; both rural Bantu tribes and urbanized, detribalized Africans undergoing acute cultural contacts in their hundreds of thousands; Coloured and Asian peoples; and White South Africans of varied national origin and all economic brackets. These apart, the facilities existing at the University of the Witwatersrand, both material and personal, as well as the programme of such researches already being carried out from this institution, would make it an obvious choice as the seat of the proposed Institute. Nevertheless, although it is proposed that the Institute be established within the framework of the University of the Witwatersrand, a cardinal aim would be to co-operate fully with and to extend all the Institute's experience and facilities to other universities and research institutes which pursue similar research objectives. The Institute would thus come to assume a national character, in much the same way as other specialized research institutes at various South African universities.

From what has been said, one of the foundations upon which Institute would rest should be *research*. Twofold in character, research would be both in the

laboratory on the accumulated materials, and in the field. It is envisaged that the Institute would promote a series of multi-purpose field expeditions, in which the combined efforts of physical and social anthropologists, linguists and ethnomusicologists, medical and dental scientists, and others, would be pooled. It is hoped that the establishment of the Institute and the inauguration of its research programmes would attract many more young men and women into these vital fields than is the case at present. In this way, personnel trained in field-survey methods would be produced, to the benefit not only of South Africa, but also of the African continent, which is sorely in need of such investigators everywhere south of the Sahara.

Aside from research, there are two other basic functions of the Institute, viz. *education* and *conservation*. The educational function would be both an intra-mural one (in relation to students and trainees), and extra-mural (in relation to the public). Conducted tours of the Museum for high school pupils and other organized groups, recitals of African music, films and illustrated talks, would all help to educate the public in the problems of Africa and her peoples. The possible integration of the Institute with the proposed civic developments in the Braamfontein area of Johannesburg might lead the Institute to be seen as a great civic asset, in the fulness of time to become a national prestige point.

The third function of the Institute would be to serve as a museum and conservatory for Africana, especially those material objects, weapons and instruments, and recordings, which over large areas of the continent are rapidly vanishing before the onslaught of detribalizing and civilizing influences. These objects and symbols need to be protected, enshrined and perpetuated, before it is too late. Already several large and valuable collections exist on the Witwatersrand and the nucleus of a museum is at hand without much searching for and amassing of new materials. To give one example: in the Department of Anatomy at the University of the Witwatersrand, is a unique collection, built up over 30 years by Professor Dart and his staff, of 600 life-masks and 500 death-masks, representing many African tribes and races from the Cape to Cairo. Adequately displayed against a background of regional maps and illustrations, this collection alone would provide an appropriate starting-point, to which could be added the collections of Bantu cultural material, musical instruments, African music recordings, the skeletons, dental impressions, photographs, fossils and implements, disease and diet records, and many other categories of objects. Finally, the museum should serve as a clearing-house for information, statistics and publications in all the fields which it will handle.

The project is an ambitious one, but the organizers believe it is not out of proportion to the stature of the man whom it will commemorate—and certainly it is in keeping with the magnitude of the problems of Man in Africa. Already the proposal has received the support of the Medical Graduates' Association, Convocation, the Students' Dental and Medical Councils, as well as of a number of professors and staff members most closely concerned.

As a first step towards the realization of this objective, it is proposed to raise the sum of £10,000 from past and present students and colleagues of Professor Dart. Once this sum is to hand, the appeal will be extended to the large financial trusts and research foundations from whom it is hoped that the greater part of the funds will be forthcoming. I would like to take advantage of the hospitality of your columns to issue an appeal to medical practitioners throughout South Africa and elsewhere to see in this proposed Institute a project worthy of their support and to contribute accordingly. Cheques should be made payable to the Raymond Dart Institute Fund and should be sent to the Medical Graduates' Association, 14a Kotze Street, Hospital Hill, Johannesburg. Bankers' stop order forms are obtainable on request from the same address.

Phillip V. Tobias,
Chairman, Committee to Establish an Institute for the Study of Man in Africa.

Medical School,
University of the Witwatersrand,
Johannesburg.

RADIO-ISOTOPES IN MEDICINE

To the Editor: We were very interested to see your Editorial comment on the growing importance of the role of radio-active isotopes in modern medical diagnosis and treatment and in your reference to the fact that the pharmaceutical industry is now processing isotopes for medical use in the course of its normal activities.

Members of the medical profession in South Africa will probably be interested to know that Abbott Laboratories (the first pharmaceutical firm to process isotopes for medical diagnosis and therapy) has recently expanded its Oak Ridge radio-pharmaceutical plant to over three times its original capacity to keep even with current demands and to expand its services to the medical profession.

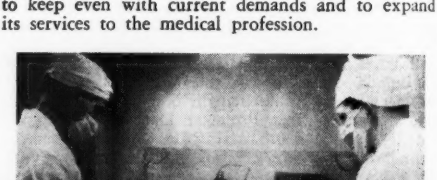


Fig. 1. High levels of radio-activity necessitate handling isotopes by remote control in a specialized 'hot' laboratory at the Abbott Oak Ridge plant, where crude radio-isotopes are converted to pharmaceutical forms and compounds for medical use.

The accompanying photograph (Fig. 1) illustrates how high levels of radio-active isotopes are handled by remote control in a specialized 'hot' laboratory at our Oak Ridge plant where crude radio-isotopes are converted to pharmaceutical forms and compounds for medical uses.

H. H. David,
Director.

Abbott Laboratories S.A. (Pty.) Ltd.,
P.O. Box 1616,
Johannesburg.